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MEDICAL SOCIETY

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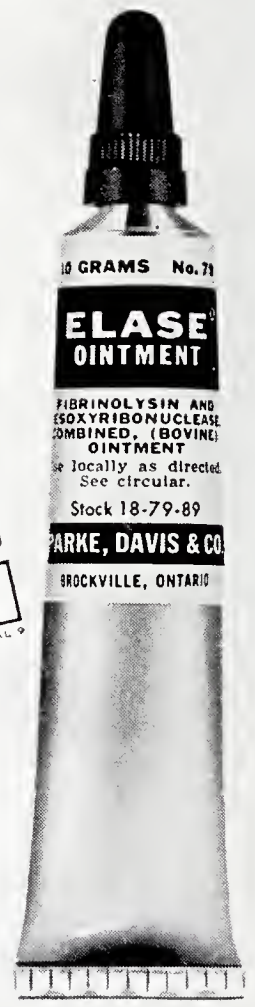

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Index

Volume XLI
Number 6
April 1964

EDITORIAL 225

FINAL REPORT

THE FIRST BREATH

ARTICLES

CHRONIC INTESTINAL PROBLEMS ..... *R. T. Manktelow,* 6T4 227

THE BATTERED CHILD SYNDROME ..... *James E. B. Miller* 6T4 231

THE JUVENILE DIABETIC ..... *H. H. Dyck* 6T4 237

JUVENILE RHEUMATOID DISEASE ..... *Mel Petersiel* 6T5 241

ACNE VULGARIS — A TEENAGE PROBLEM
..... *Edward G. Davis* 6T4 245

TWO HIP CONDITIONS COMMON IN CHILDHOOD
..... *Ronald B. Fildernan* 6T4 247

MATURATINAL LAG ..... *K. Whytock* 6T4 249

FEATURES

APOLOGY 229

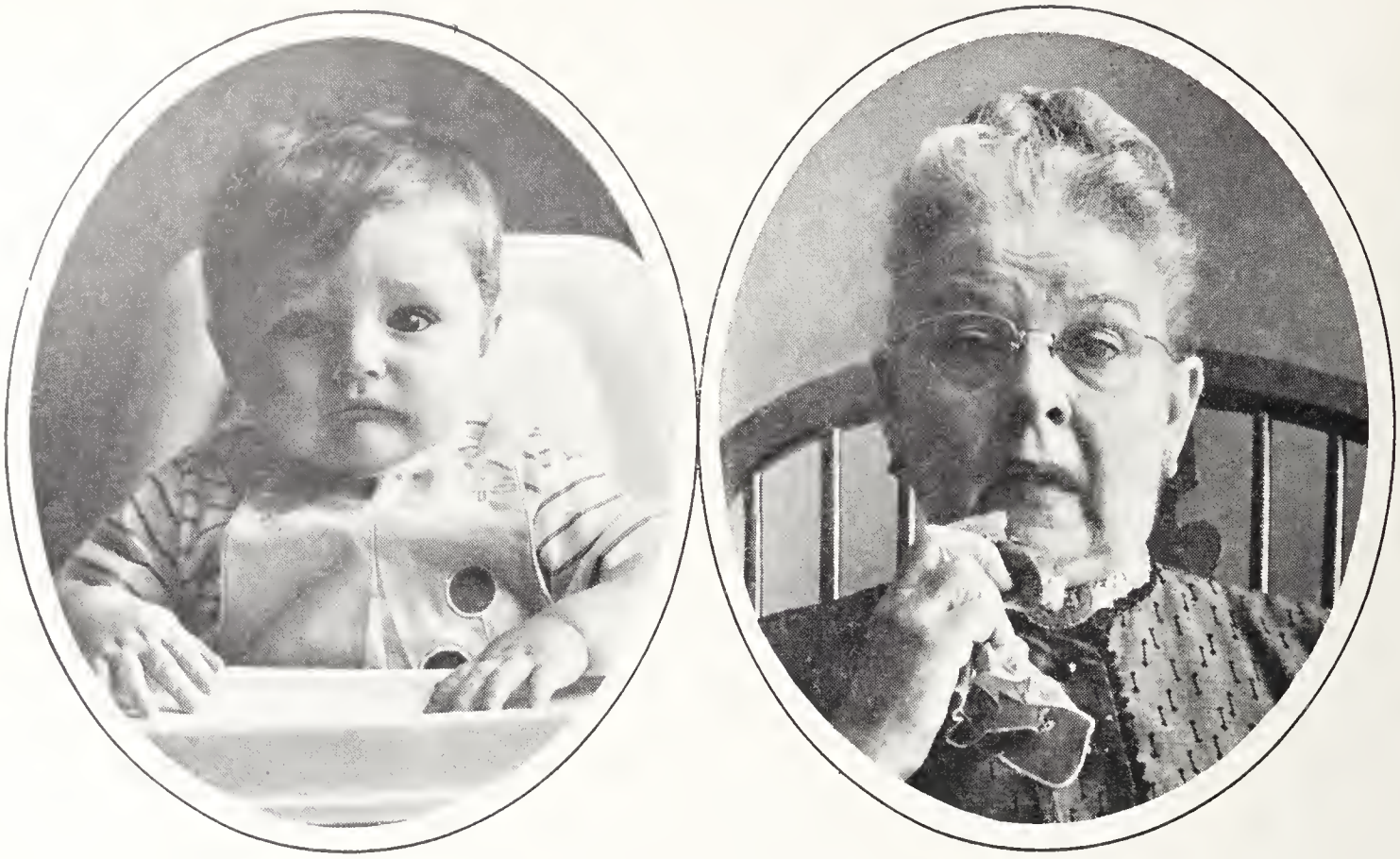
RECENT ADVANCES 230

CORRECTION 246

BOOK REVIEW 254

INDEX TO ADVERTISERS

PARKE DAVIS ..... Inside Front Cover
ORTHO ..... Inside Back Cover
POULENC ..... Outside Back Cover
SMITH, KLINE & FRENCH 219
FROSST 222
ABBOTT LABORATORIES 224
SEARLE 226
PITMAN-MOORE, DIVISION OF DOW CHEMICAL OF CANADA 240
HOMEWOOD SANTARIUM 252
TAYLOR INSTRUMENTS 252 253
UNIVERSITY OF TORONTO BOOK STORE 254
OSBORNE'S PHARMACY 254



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Editorials

FINAL REPORT

When I began to write this editorial, it was my hope to find a very succinct quotation that would express the mixed feelings experienced during the past year as a co-editor-in-chief: those of frustration, anxiety, and pride. My search proved to be impossible.

My co-editor, Fred Langer, and I are deeply chagrined that we have been unable to meet production deadlines. The experience has been most embarrassing and yet, quite enlightening. It is our hope that other undergraduates of the faculty appreciate the degree to which they added to our tardiness.

We feel that the editorial material in the six issues of volume XLI has, on the whole, been of a very high quality. It is a fact that more people have endeavoured to write for this set of journals than ever before. This particular fact has been most gratifying.

We want to thank all the individuals that have so ably managed the business affairs of the journal. Without their most appreciated efforts, it would be impossible to publish a journal, even a tardy one.

Next year, there is again a dual editorship. The two new editors are J. S. Burke and B. E. Koehler, both of next year's graduating class. It is my personal belief that two editors are needed in order to divide the work load into a reasonable number of hours. We wish the new editors every possible success.

Wm. S.C.

THE FIRST BREATH

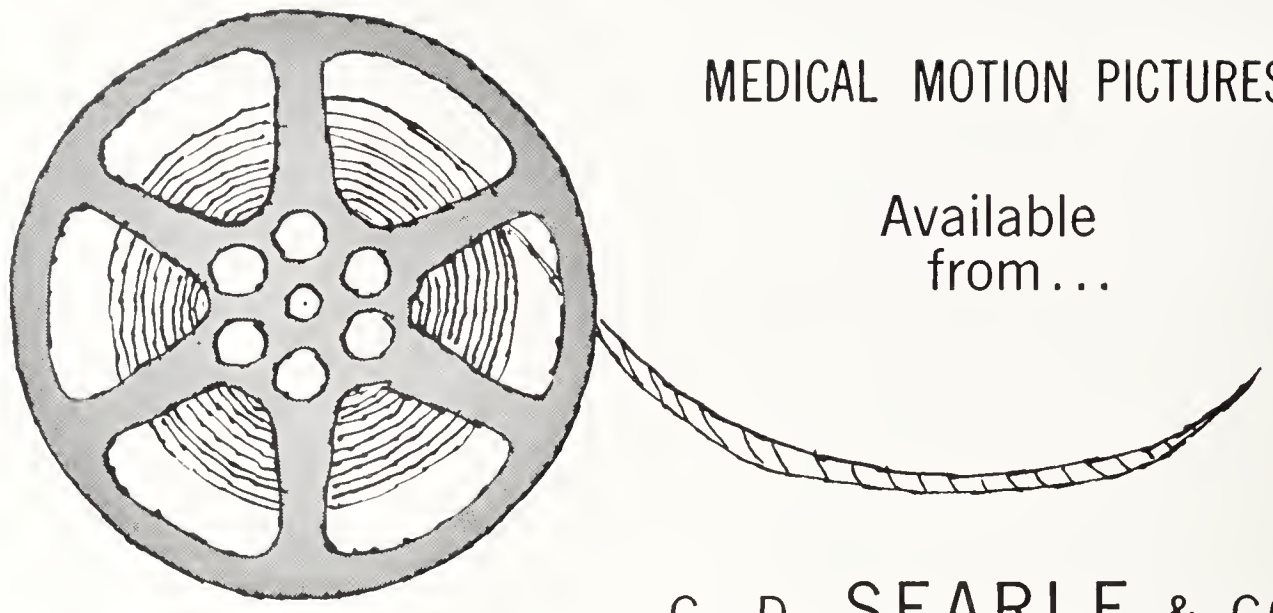
Most medical students can still remember seeing their first delivery, and tensely waiting for the infant to take its first breath. After a while, this phenomena became passé, and became accepted like many of the little understood dramas of normal physiology.

What is known about this first breath? How does the baby initiate the act, and what force is required? It is surprisingly hard to answer these questions. We are aware that the levels of carbon dioxide, oxygen and acidity, regulate the breathing of adults. Presumably, one of these stimuli is active in initiating the breathing in infants. Upon delivery, the infant is suddenly thrust into an unfamiliar world, and assailed with the stimuli of touch and temperature change, light and sound. It is likely that these physical stimuli also play a part in initiating breathing.

Apparently, at the moment of delivery, the infant's lungs are not collapsed as formerly thought, but contain fluid. Thus there is presented to the first breath the combined resistance of surface tension and viscosity. It is probable, that this prior filling of the airways may provide less surface tension than if the walls of the duct were collapsed and adjacent.

At the outset, the baby's chest is shaped somewhat like a cone. The essential force for inspiration, comes from contraction of the diaphram, which flattens and descends. Measurement of the drop in intrapleural pressure during the first breath has revealed some surprising results. It appears that the normal baby may require a negative pressure of 20 to 30 cm. of water. And, in situations of obstruction, the baby has a capacity to create a negative pressure of at least 70 cm. of water. It is believed that many breaths, each having a volume of 15 to 30 cm. are necessay, to create the whole lung. During these breaths, the liquid in the airways is gradually absorbed by the pulmonary capillaries. The practical implication of these facts is that considerable pressure may be needed in the first few efforts at artificial resuscitation if the baby has made no effort to breath.

R.T.M.



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Chronic Intestinal Problems

R. T. Manktelow, B.A., 6T4

In recent years, considerable interest has developed in the field of intestinal diseases in children. The discovery of several specific disease entities, with the elucidation of definite criteria for their diagnosis, has done much to stimulate this interest.

It is proposed to discuss four separate diseases, each of which involves the intestinal tract in childhood. Gluten induced enteropathy, cystic fibrosis, disaccharide intolerance and primary protein losing enteropathy have been selected for discussion.

Celiac Disease
(Gluten Induced Enteropathy)

In certain children, the ingestion of foods containing wheat and rye damages the intestinal mucosa. The offending substance in these grains is contained in their gluten fraction. A decrease occurs in the absorbing capacity of the mucosal surface, involving all foodstuffs including fats, protein, carbohydrates, vitamins and minerals.

The affected child produces a stool which is greasy, pale, bulky, frothy, foul smelling, and floats. The child shows signs and symptoms of malnutrition and a distended abdomen. Actually, Samuel Gee described a similar clinical picture in 1888 and named it the Celiac Affection, or celiac syndrome, as we have come to call it. The sine qua non of this syndrome is an increased excretion of fecal fat. It is only in recent years that gluten intolerance has been recognized as one of the important causes of the celiac syndrome. Unnecessary confusion has resulted from the use of the term celiac disease to describe gluten induced enteropathy (G.I.E.) Cystic fibrosis, which is the commonest cause of the celiac syndrome, will be discussed in the next section.

Typically, the infant with gluten induced enteropathy is normal at birth. Shortly after the introduction of gluten containing foods to the diet there is a gradual development of the symptoms and signs of the celiac syndrome. In addition, the infant becomes irritable and severely anorexic. Eventually, a variety of findings may develop secondary to protein, vitamin, or iron deficiencies.

Because of the availability of specific but rather cumbersome dietary therapy, the diagnosis should be confirmed by certain investigative procedures. The first step, if possible, should be the demonstration of steatorrhea suggested by the pale greasy stools. Fecal fat losses averaged for 3-5 days on a routine diet should exceed the normal value of 5 gms/day. Carefully carried out glucose and vitamin A tolerance tests give a fair reflection of the capacity of the intestines for absorption of CHO and fat. In the child with GIE both of these should be flat. The development of a technique for peroral biopsy of the intestinal mucosa has contributed greatly to an understanding of intestinal function and disease. The findings which are characteristic of GIE are shortened, flattened villi, damaged surface epithelium and round cell infiltration of the lamina propria. To date, no child has been described with such a pathological picture in the small intestine who has not responded to removal of gluten from the diet.

The treatment consists of a diet which excludes all gluten containing foods. Because of the variety of processed foods which contain wheat and rye, the novitiate must procure a list of gluten containing foods. The acutely ill child may require intravenous therapy for a few days until the intestinal tract is settled. A diet free of gluten can then be started. In the early stages of therapy, it has been found beneficial to keep the diet low in fat and high in protein.

The criteria for the success of the diet, which in a sense are confirmation of the diagnosis, are quite specific. Within one to two weeks the child will change from being irritable and cranky to become happy and contented. The fat content of the stool should return gradually to normal. Weight gain and increase in linear growth may be dramatic. Occasionally the improvement may be slow, and the child may need a trial of 6 months before the diet should be abandoned. If a complete response is not obtained one should look for associated complications such as enteral or parenteral infection or iron deficiency anemia. Occasionally it may be necessary to remove (from the diet) cow's milk, which may act as an additional irritant to the intestine.

Most authorities feel that dietary control should be maintained until the end of the child's growth period. Resumption of a gluten containing diet may cause no signs or symptoms except for the insidious inhibition of linear growth.

In final comment, it should be mentioned that mild uncomplicated malabsorption is not uncommon. Recurrent episodes of celiac stools may occur, particular-

ly in the 1-4 year age group. However, laboratory investigations are negative. An uncommon disease (incidence 1:2000 - 1:6000), GIE is considered as a diagnostic possibility in many cases of chronic diarrhea or growth failure. With modern techniques an accurate diagnosis can usually be made and effective therapy initiated.

Cystic Fibrosis

This disease is an autosomal recessive that affects all races to some degree. The incidence in western civilization is 1:1000 births.

The clinical and pathological manifestations of cystic fibrosis are the result of the abnormal mucous formation and altered electrolyte control in the exocrine glands throughout the body. Clinically, the respiratory and gastrointestinal systems are the ones, most severely affected by the disease. Inspissation of mucous secretions within the pancreas blocks off the secretions of this gland from reaching the intestines. As a result of being without trypsin, lipase and amylase, intestinal digestion is severely impaired.

Consequently, these infants often have the type of stool associated with the celiac syndrome (pale, foul, bulky, floating on water). Frequently the child is brought to the doctor because of repeated or protracted chest infections. They are usually of low birth weight, and may have clinical manifestations of the disease at birth. Not infrequently, they do not get into serious trouble until late infancy or childhood. The mother may comment that the child "tastes" salty, due to the abnormally high concentration of electrolytes in the sweat. Frequently, there is a family history of death in childhood or infancy, which was unexplained, or labelled chronic diarrhea. The important findings on examination are an emphysematous chest, scattered rales, clubbed fingers, a distended abdomen and signs of malnutrition. Usually the child is not irritable and has a good appetite, in contrast with the child with GIE.

A number of tests may be employed to confirm the diagnosis. The most useful is a measure of sweat chloride concentration, the normal value of which is below 60 mEg/I.

The chest x-ray shows a variety of changes, chief of which are overaeration, increased peribronchial infiltration and, in the acute case, patchy bronchopneumonia.

The condition of the pancreatic enzymes is most satisfactorily determined by tolerance tests. The glucose tolerance test should be normal, unless there is hepatic or pancreatic islet dysfunction. The fat soluble vitamin A tolerance test should be flat because of the absence of lipase. In the light of these findings, duodenal intubation for the search for pancreatic enzymes is usually unnecessary.

One should be aware of a number of facts concerning these tests and the altered physiology which they represent. Sweat chlorides, which are always elevated eventually, may be normal for the first year of life. Pancreatic dysfunction may be incomplete, or may, especially in the early stages, not exist at all. The blockage of the pancreatic ducts is probably a gradually progressive process. For these reasons the diagnosis is occasionally difficult to make in the first year of life.

In summary, the diagnosis can be made in the presence of elevated sweat chlorides, and either abnormal bowel function, or recurrent chest infections, or both.

The disease is incurable. The intestinal complement of the disease is not usually the limiting factor to life and is relatively easy to manage. In order to replace the missing pancreatic enzymes, commercially prepared pancreatic enzyme is administered. The enzyme is sprinkled on the food, or eaten separately with every meal and every snack. The correct dose of enzyme is the amount necessary to return the stools to normal. Extra vitamins and table salt should be added to an otherwise normal diet.

Death is usually due to acute or chronic chest infection. The successful management of the respiratory routine depends upon considerable experience and care. Treatment must continue for the life of the patient.

Disaccharide Splitting Enzyme Deficiencies

The disaccharides important to human nutrition are maltose, lactose and sucrose. They must be ingested as such or reach the small intestine as the result of the digestion of poly-saccharides (e.g. starch). In the process of absorption the disaccharides are split into their component monosaccharides in the mucosal cell of the small intestine The disaccharidases involved mal (maltase, lactase, and invertase) are now known to be concentrated in the mucosal epithelium, and not the succus entericus.

Infants with the congenital absence of one or more of these enzymes have now been described. If the enzyme is missing, then its corresponding disacchaside will not be broken down and absorbed, but will remain in the lumen of the intestine. The disaccharide may then be attacked by intestinal bacteria. Organic

acids such as lactic and acetic acid are formed. These acids irritate the intestinal wall, causing increased secretion of fluid and mucous and increased persistalsis. As a result the infant has foamy diarrhea. In infants with lactase deficiency, diarrhea develops soon after birth, particularly if the baby is breast fed. In others, the foamy diarrhea occurs immediately after the offending sugar is added to the diet.

In the absence of feeding with breast, lactic acid or protein milk, the diagnosis can be suspected on finding a low stool ph, due to high levels of organic acid. Further confirmation can be gained from a tolerance test with the appropriate disaccharide. In some centres, enzyme assay can be done on tissue obtained from intestinal mucosal biopsies.

Up to the present, we have considered only absolute deficiencies. However, there is evidence that the deficiency may in some cases be partial, in which case the child will grow out of it. The characteristic stools are formed only if sufficient disaccharide is taken to overload the weakened enzyme system. It is possible that one of the reasons why some babies do better when removed from breast milk is a partial lactase deficiency, as there is a higher lactose concentration in breast than cow's milk.

It is felt that partial enzyme deficiencies in dystrophic malnourished children may be the explanation for their abnormal stools. Similarly, a secondary enzyme deficiency may be present after enteritis, through temporarily damaging the intestinal wall. The irritation of the fermentation products may act to maintain this damage which may only be relieved when the sugars are temporarily removed from the diet.

The treatment for a specific enzyme deficiency is the removal of the offending sugar from the diet or the addition of the missing enzyme to the diet.

Primary Protein Losing Enteropathy

Protein losing enteropathy involves all those disease states which incur leakage of plasma proteins into the gastrointestinal tract. This protein loss may be a manifestation of many disease states, most of which involve the intestinal wall. Hypoproteinemia is the result as both albumin and globulin are depressed. Some patients have a protein losing enteropathy that is not associated with any known intestinal disease. These patients are said to have primary or idiopathic protein losing enteropathy.

Protein leakage into the gastrointestinal lumen occurs as a significant mechanism of plasma protein degradation in the normal state, and accounts for between 25-50% of the daily turnover (degradation) of serum albumen. The protein lost into the intestine is subject to the normal digestive processes and hydrolysed into amino acids and reabsorbed. Thus, the degree of depletion of total body protein will be dependent not only upon the quantity of leakage but upon the efficiency of reabsorption and resynthesis. Whether or not the excessive loss observed in intestinal diseases is a simple extension of processes normally operative has not been determined.

In primary protein losing enteropathy, peroral biopsy may reveal marked dilatation of lacteals and lymphatic spaces, together with some degree of edema of the lamina propria. The major clinical findings are a non-specific diarrhea and dependent edema. A malabsorption state may occur.

At present, no definitive therapy is available for protein losing enteropathy. The management of edema with salt restriction and diuretics constitutes the mainstay of symptomatic treatment.

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APOLOGY

The editors wish to apologize for not including the articles of E. G. GOH and J. Y. WAKIL of 6T4 and that of B. RAXLEN of 6T5. These articles, particularly the latter, were all of high quality; however, economic realities forced us to reduce the editorial content. It is our hope that these articles will be published next year. *Wm. S.C.*

Recent Advances

The Perceptually Disturbed Child

About 12-17% of the public school population exhibit learning difficulties in some form. There is a tendency in both teachers and parents to simply place many of these children in the classification of "stupidity of unknown etiology" and attempt no treatment at all. Even if such a child is investigated, often he is categorized under his major defect, while ignoring other symptoms present. Thus, the child is approached as if he were his label, "the emotionally disturbed child" or "the retarded child". Such an approach can never provide an adequate basis for effective remedial programme. Rather the overall development of the child must be considered, and his potential evaluated according to the results. A child must be considered as a dynamic organism, capable of change and progress, rather than a static entity with an unalterable label.

Only about 2% of all public school children exhibit generalized learning difficulty; that is, learning difficulty which may be attributed to a retardation in intellectual development. On the other hand, 10-15% of all children show specific learning difficulties of one kind or another (although most of these are in reading). Therefore, to investigate learning difficulties, one must also consider areas of the child's physical and mental growth other than intellectual development. The other areas which have been found to contribute most significantly are those of motor development, perceptual development· language, emotional and social development.

Possibly the most frequent cause of learning difficulties (and, at present, the least recognized) is that of a disturbance of the child's perceptual abilities — visual, auditory, or kinesthetic. Perceptual disturbances do not necessarily imply defects only in the external sense organs but may involve faulty interpretation of sensory data by the brain. In order to adequately understand these disabilities, it is first necessary to have a general idea of the theory of perception. According to Kephart, the perceptual process begins with the stimulation of a sense organ, which in turn sends a pattern of neural impulses to the sensory cerebral cortex. The impulses are then conducted, via internuncial neurones into the surrounding association areas where the integrative process takes place. This involves the integration of the impulses from all the sensory organs, and the coordination of past experiences with the present information. Thus, a highly complex pattern is elaborated; this is scanned by a scanning device and translated into an output pattern. Enroute to the muscle groups a portion of the output is drained off and fed back into the system at the input end, where it is used for control. If the muscles do not respond properly, this is reflected in the feedback. Any portion of this mechanism may be disrupted in the perceptually disturbed child, but the only accessible parts are those of input and output. In any treatment the general approach must be that of attempting to stabilize the output (sufficient motor control to permit stable motor responses) and the input (control by the child of the information given to him by his external sense organs), and finally, to encourage the child to behave in terms of these two functions at once (using vision as an example, to reach out and touch an object, and to watch the process and control it by visual stimulation).

Because of the many factors which may be affecting the child with learning difficulty, it is important to assess the child both qualitatively and quantitatively. Even when perceptual difficulties are implicated, there are still the three possibilities of visual, auditory, or kinesthetic. Frostig has evolved a method of testing visual perception, by which a child's perceptual quotient and perceptual age equivalent can be obtained. The child is required to attempt carefully graded tasks in five areas of visual perception:

1. Eye-hand coordination — drawing straight lines between increasingly narrow boundaries;
2. Figure-ground perception — discrimination between intersecting lines;
3. Perception of form constancy — detection of squares, circles among other shapes on the page;
4. Perception of position in space — detection of a reversed or rotated figure in sequence;
5. Perception of spatial relationships — copying patterns by linking dots.

It has been found that intercorrelations among the five subtests are low. A clear connection can be found between disabilities in visual perception and poor classroom adjustment at the lower age levels, thus suggesting that defects in visual-perceptual processes may affect the development of the child 'in toto'. At this time, however, the test is of no use in establishing the etiology of the perceptual disability, be it brain injury, developmental lag, or emotional disturbance.

(see page 244)

The Battered Child Syndrome

James E. B. Miller, 6T4.

Introduction

"The Battered Child Syndrome" is a term used to characterize a clinical condition in young children who have received serious physical abuse and/or neglect generally from a parent, foster parent, or older sibling. It is derived from the fact that the child's injuries are the result of twisting, throwing, knocking around or some other form of "battering" by the abusive person. The condition has also been described as "unrecognized trauma to the skeleton or traumatic periostitis". The fact that permanent injury resulting in future disability is frequently involved in this syndrome is ample justification for its importance. Moreover, injuries may be so extensive and of such a serious nature that the child's clinical recovery will be terminated by death.

Child abuse and neglect are not new in our society. Social agencies have struggled with these problems for years. However, what is new is the increasing awareness of this problem over the past six years not only in the medical profession, but also in the legal field as well as the lay press. Although there is an increasing awareness of these commissive acts of physical abuse, the topic is still new and unknown to many of us.

In order to make a complete coverage of the BCS, it should be kept in mind that the problem of the parentally mistreated child involves more than acts of commission. There are also acts of omission whereby the child is deprived of adequate nutrition. This is frequently referred to as the "nutritional variant of the BCS" and not only is equally (and probably more) dangerous to the child's welfare, but also is equally censurable under the law. The main discussion in this presentation, however, will be limited to that of the physically battered child, but some reference will be made to the equally important nutritional variant.

Clinical Manifestations

These vary widely from those cases in which the trauma is often so mild that it is unsuspected and unrecognized to those who exhibit the most florid evidence of injury to the soft tissues and skeleton. In the former, the etiology is often misdiagnosed as failure to thrive from some other cause or as arising from a metabolic disorder, an infectious process, or some other disturbance. In such patients, specific findings of trauma (e.g. bruises or visible x-ray changes) are frequently misinterpreted or their significance not recognized.

The following chart, listing the chief complaints in seventy-two proven cases of the BCS illustrates the many ways in which it can present itself.

Disability of Extremity:		
—injury to one extremity	12	
—weakness, pain, swelling of one extremity (without history of trauma)	13	25
Skin Lesions:		
—bruises, petechial hemorrhages	12	
—impetigo	1	16
—"lumps"	2	
—swelling of the face	1	
Failure to Thrive:		12
Convulsions		12
Dehydration from Vomiting and Diarrhoea		4
Macrocephaly (due to Subdural Hematoma)		3
Total Cases:		72

This syndrome may occur at any age in childhood, but generally it falls within the age bracket between birth and three years of age with the peak incidence at three months. (In contrast to this, the age incidence of accidents in children is minimal below the age of nine months.) Frequently, there may be only a single episode of trauma, but more often there are multiple soft tissue and skeletal injuries associated with poor general health and neglect including poor skin hygiene and malnutrition. Several features of this syndrone, which alone are not diagnostic but must be accompanied by extensive investigation of the child's environment in order to make a diagnosis, are as follows:

1. There is a marked discrepancy between clinical findings and historical data as supplied by the person in charge of the child.

2. There are no new soft tissues or bone lesions while the child is in the hospital or in a protected environment and this tends to exclude many diseases of the haemopoietic and skeletal systems in which lesions may occur spontaneously.

3. There are often multiple bony lesions, either in one bone or several at various stages of healing.

4. There is a frequent association of subdural haematoma with multiple skeletal injuries.

5. Distribution of lesions in the abused child is unrelated to rates of growth. Moreover, an extensive lesion may be present at the slow-growing end of a bone which otherwise is normally mineralized and shows no evidence of metabolic disorder at its rapidly growing end.

6. In addition to manifestations of physical abuse, there may also be evidence that the parent or parental substitute has also assaulted the child by administering an overdose of a drug or exposing the child to natural gas or other toxic substances.

Radiologic Features

Radiologic examination plays two main roles in the problem of child abuse. Initially it is used as a tool for diagnosis. Subsequently, it is valuable as a guide in management not only to follow the progress of healing, but also to detect evidence of new injury. The x-ray signs previously discussed result from a combination of circumstances.

(a) *Age*: As a general rule, the children are less than three years of age during which time the relative amount of radiolucent cartilage is great. Therefore, anatomical disruptions of cartilage without gross deformity are radiologically invisible or difficult to demonstrate. However, since the periosteum is less securely attached than in older children and adults, it is therefore more easily and extensively stripped from the shaft by massive periosteal haematomas following trauma. Subsequently, new bone formation can take place around and remote from the actual fracture site and is visible on x-ray.

(b) *Nature of Injury*: Injuries to the appendicular skeleton are the most common in this syndrome due to the fact that the extremities are the "handles" for rough handling. The forces applied by the adult hand in grasping and in seizing usually involve traction and torsion which are the forces most likely to produce epiphyseal separation and periosteal shearing. However, such forces may be innocently applied as in grasping the child's arm to prevent him from falling or suspending him by his feet to dislodge a foreign body caught in his throat. But again evidence of repeated application of such forces should arouse suspicion of underprotection if not of aggressive abuse and lead to extensive investigation to ascertain what really happened to the child. Shaft fractures, on the other hand, result from direct blows or from bending or compressive forces.

(c) *Length of Time between Injury and X-Ray*: No signs of bone injury are found during the first week after injury unless gross fractures or dislocations were produced. Reparative changes, which are seen on x-ray as excessive new bone reactions, may become manifest about twelve to fourteen days post-injury and can increase over the subsequent weeks depending both on the extent of the initial injury and the degree of repetition of further injury. It is important to note that new bone reaction can be confused with neoplastic change.

(d) *Repetition of Injury*: This is an important factor in producing diagnostic radiologic signs of the syndrome. Evidence of several bony lesions either in one particular bone or in several different bones and appearing at various stages of healing is always indicative of repetitive injury. While one isolated injury may reasonably be attributed to a natural accident to which a young child is so susceptible, it is less plausible to attribute a series of injuries to a series of "natural" accidents in the same individual in a carefully supervised environment. Moreover, since the majority of children found to have multiple injuries are approximately three months of age, then these injuries could not have resulted from unsure attempts to walk or from climbing into hazardous areas. Under such circumstances, an environmental factor must be considered. It is of interest to note that there are no differences in the x-ray appearance of the individual lesions which distinguish multiple skeletal injuries in late infancy and childhood from those incurred at time of delivery. Thus to exclude the latter cases, there must be a serious clinical judgement made, based on a careful assessment of the child's birth history.

Utilizing x-ray techniques as the only means of investigating the case of "a battered child" is not without danger as x-rays do have obvious shortcomings. The dependence on x-rays immediately after injury as a chief criterion of abuse may result in overlooking some cases because bony changes, unless gross, only appear radiologically as signs of repair some twelve to fourteen days after injury. In addition, the x-ray will depict only the condition of the bones and not the train of events leading to the child's injury(ies).

Differential Diagnosis

Although radiological signs of the BCS may be quite characteristic, it is generally found that other diseases are considered because of the reluctance to accept the implications arising from the conclusion

that the bony lesions result from battering. Without evidence of obvious fracture or knowledge of injury, the bony changes may be diagnosed as sequelae of scurvy, syphilis, chronic vitamin A poisoning (hypervitaminosis A), infantile cortical hyperostosis, or osteogenesis imperfecta. The following is a brief discussion of these diseases:

Scurvy: A systemic disease with all the bones presenting a generalized osteoporosis and exhibiting large calcifying subperiosteal hemorrhages if some treatment has been undertaken, which may extend the entire length of the shaft but appear especially in areas of rapid growth. In addition, the white line of Fraenkel (representing the zone of well calcified cartilage at the metaphysis) and the Halo sign (representing the epiphyseal centres of ossification surrounded by a white ring) the scorbutic rosary and the lower limbs in the "frog position" are characteristic of scurvy and not trauma. Similarly, if the patient's "true" dietary history has been obtained, it will reveal evidence of vitamin C deficiency.

Syphilis: In the first months of life, this disease can present with metaphyseal and periosteal lesions. These bony lesions tend to be symmetrical and are usually accompanied by other manifestations of the disease (e.g. rhinitis, with or without blood followed by a maculopapular or morbilliform rash especially on the back, buttocks, and on the backs of the thighs. Also, anaemia, hepatosplenomegaly or jaundice may be present.)

Hypervitaminosis A: This presents with cortical hyperostosis which appears as wavy lines along the shaft of a tubular bone but does not extend to the metaphyseal or epiphyseal ossification centres. Anorexia, irritibility, itching associated with hard, tender lumps on the extremities, fissures of the lips, loss of hair, hepatomegaly and jaundice are part of the clinical picture.

Osteogenesis Imperfecta: The bony changes here are generalized and are seen in bones other than those involved in the area of trauma. In this disease the fractures are commonly of the shaft; whereas, in the BCS they usually occur in the metaphyseal regions. Moreover, the typical mosaic ossification pattern of the cranial vault in this disease is not seen in the abused child even though skull fractures are present. Also, characteristic of this disease are the bright blue sclerae which obviously do not result from trauma.

Infantile Cortical Hyperostosis (Roske-Caffey-Smyth Syndrome): A benign disease of unknown etiology usually appearing at two months and never greater than five months. It usually involves the mandible with a lower incidence in the clavicle, ulna, humerus and ribs. The periosteal changes may extend to muscles and fascia. The diaphyseal lesions seen in this condition serve to differentiate it from the metaphyseal lesions of the abused child.

Pathogenesis

What can possibly explain the brutal attacks on these defenseless children? It has long been recognized that child-rearing patterns both good and bad are passed from one generation to the next. This practice is acceptable and encouraged insofar as the good patterns are concerned. But for those parents who were brutally treated in their childhood and adolescent years and who intend to do likewise with their own children the practice is undesirable. It can only result in broken families and battered children.

An unwanted child can very readily become a target for excessive physical abuse. Such a child may have resulted from a premarital conception and either one or both of the parents forced into this marriage may choose to beat the child as punishment for his causing their unhappy and unsuccessful union. On the other hand, the pregnancy may have occurred too soon after the marriage to allow the couple time to adjust to problems of married life or at some other time when a new addition to the family was not planned.

Often the sex of the child attacked is related to specific factors in the composition of the abusive person's neurosis. It is also interesting to note that often only one child in the family receives abuse; whereas, the remainder of the family receive loving care. Under these circumstances, it is quite possible that the abused child has become some sort of symbol to the parent and the parent reacts to this symbol by attacking it. In addition, the young child may be attacked in a fit of jealously by an older sibling. The latter is usually an over-demanding child who was the centre of attraction before the younger sibling's birth. He then attacked the infant in an effort to relieve his frustrations of no longer being the centre of family life.

The type and degree of attack may vary. At one extreme, the attack is by a frank psychotic and the resultant injuries are so serious and extensive that death follows, while at the other extreme, overzealous spanking and slapping occur but no overt harm is done. Between the two extremes there are a large variety of cases. In some cases, the abusive person was found to be of very low intelligence and to have difficulty in controlling his aggressive behaviour. Fre-

quently, the child was provoked into behaving in a manner previously judged to be undesirable by the abusive person who then felt justified to beat the child as punishment for his actions. Often the abusers were described as psychopathic or sociopathic personalities frequently associated with alcoholism, sexual promiscuity, unstable marriages and minor criminal activities. In addition, they were usually immature, impulsive, self-centred, hypersensitive and quick to react with poorly controlled aggression.

Child-beating may also occur among people with good education and stable financial background when some defect in character structure leads to uncontrolled expression of aggressive impulses. Here too, there may be some suggestion that the abusive person was subjected to similar abuse in childhood. Under these circumstances, the person is identifying himself with the aggressive parent, but the identification occurs despite the strong wishes of the person to be different.

Management

The pathogenesis in each case of the Battered Child will of course be very important in determining the course of management. The principle concern here is to make an early and correct diagnosis. The attending physician should then contact the appropriate social agencies and/or legal authorities in order to ensure adequate protection for the child and to carry out a thorough investigation of the child's environment in an effort to determine the cause of his injuries.

Problems in Management

The proper handling of a case involving a battered child is fraught with many difficulties. As mentioned previously, few clinical symptoms can be directly co-related with a physically abused child. As a result, it is impossible to even attempt to establish a classical description that would represent all cases and thus simplify clinical diagnosis. To make the diagnosis more difficult is the fact that purposeful physical abuse by any person (be he parent, guardian, sibling or other person having permanent or temporary custody of the child) will ordinarily be committed in the home where the sole witnesses are members of the immediate family. In a situation such as this, neither will the abuser readily admit to these abusive practices nor will the witnesses bear evidence against the abuser because they both fear the implications of public and legal censure upon themselves and their family. Furthermore, if the abused child is too young to speak, then there will be no complainant and the abusive action will remain undetected. But, if the child is old enough to speak for himself, not only will listeners come to conclusions based on subjective feelings rather than seeking out the true facts, but also the credibility of the story itself will be based upon the degree of the maturity and extent of his imagination. Detection is also made more difficult in some cases by the parent(s) dispersing evidence by taking the child on different occasions to different doctors or hospitals.

After the suspicions of the attending physician have been aroused regarding the true nature of the child's injuries, he should report the cases to the appropriate agencies (and/or higher authorities if necessary). It is the duty of the physician to acquaint himself with the facilities available in private and public agencies and therefore will be able to contact the appropriate agency which will be most useful in assisting with the problem at hand. Before the subsequent investigation of the child's home environment takes place, it is best to arrange for the child to be removed from the home in order to prevent further injury. Any of the agencies contacted may be of assistance in bringing the case before the court which alone has legal power to arrange for temporary or permanent separation of the child from the parent's custody and his placement with a responsible relative or well-supervised foster home. The investigation itself should include an assessment of the home situation, parental attitudes, the validity of the parents' explanation as to the cause of injury and the strength with which they express a desire for the child's return. The rationale of reporting is that a case of suspected child abuse constitutes a medical and social emergency and as the usual pattern is repetitive abuse, it is important that a prompt preventative action be taken.

Nevertheless, case histories from many hospitals substantiate the fact that repeated medical contacts with children suffering from possible physical abuse are made without being reported. This failure to report on the part of the physician may have several explanations. It may be due to the newness of this syndrome of multiple skeletal and soft tissue injuries occurring over a period of time, that overlooking isolated or first injuries is quite understandable. Moreover, the physician may find the idea of parents abusing their children so abhorrent that he denies the possibility that abuse might have been the cause of injury. Likewise, young physicians, internes, and residents, finding such behaviour too bizarre for belief try to explain the physical signs as manifestations of some rare disease. The physician may also hesitate to report because there may be instances where there is difficulty in determining the dividing line between punishment and outright abuse. Another reason is the attitude of neglectful doctors, although fortunately few in number, who

state that they are doing everything medically possible for the child and that no more should be expected of him. Others hide behind exaggerated fears of court procedures, adverse publicity or even of suit for false accusation. Still others are reluctant to subject a family, either well-known to him or complete strangers, to the expense, embarrassment and emotional strain of a court proceeding. Finally, many physicians fail to act because they did not know what to do.

The following is a resumé of the revised draft prepared by The Children's Bureau, Department of Health, Education and Welfare in Washington, D.C. This describes what has been suggested in the United States (at time of writing it was not known whether this draft had been made a legal document). It requires that any practicing physician who has reasonable cause to believe that a young child brought to him has had serious injury(ies) inflicted upon him other than by accidental means by the parent or other person responsible for his care, shall report this to the appropriate authorities. The draft also suggests that arrangements will be made for protection against such matters as defamation of character, malicious prosecution and any successful litigation brought against the doctor, the expense of which might be no trivial matter. Finally, some provision will be made to prosecute those who knowingly or willingly violate the rules of the draft.

No one believes that requiring doctors to report cases of suspected physical abuse will end or even lessen it. But such a law and the process of enacting and implementing it would focus attention on the problem, alert the medical profession to its incidence and serious nature and perhaps set in motion community action to provide better protective services for the children.

The attending physician himself can frequently obtain very valuable information from the person under investigation by developing a routine of careful questioning. He should, however, avoid making any accusations so as not to antagonize the person and consequently lose control over the case before constructive measures can be accomplished and perhaps create a situation where the abused child may become a target for further injury. Additional clues may be acquired by asking questions to uncover concealed sources of worry or tension and feelings of hostility toward the person's family background to determine if these had any bearing on how they treated their own child. It is more advantageous if the parents are questioned separately for a two-fold reason; (1) One parent may be away from home for long periods and therefore may be entirely unaware that the child has been abused. (2) Parents will often go to great length to protect each other by denying the partner's involvement.

Two of the references (Wooley and Evans) at first told the parents that they were aware of the family problem and offered assistance in resolving it without using punitive action. This was not too successful so they later attempted enlisting aid from social agencies. However, many families reacted to this intervention by moving away from the jurisdiction of the agency involved and often by subjecting the child to further abuse. The subsequent policy became one of arranging to have the child removed from the home by cooperative efforts (social agency and court action) before the home environment was investigated. However, if the child has been hospitalizd for the treatment of his injuries, then the investigation should be made before his discharge from the hospital.

After the initial investigation, some children may be returned home on the condition that the parents utilize the services of an agency appointed by the hospital. This provides an opportunity for the case-worker to further assess the home situation by observing the child in his customary environment including all the familial cross-currents especially the manner in which the child and parents interact. A small infant's fear and withdrawal from an intimidating and abusive parent is hard to detect except by spending hours with the parents and child.

Just as Rome was not built in a day, neither can the home environment be constructively altered over a short period of time. It takes a great deal of time and patience by all concerned in order to rectify some of the warped ideas that exist concerning child upbringing and alleviate the bitterness and hostility held against the child. If the child remains in or is returned to an unaltered environment he is exceedingly vulnerable to further injury. Thus separation either of the child or of the abusive person from the family needs to be undertaken. Even when the person is in need of and accepts psychiatric help, separation is still necessary during the period of treatment. The problem of separation usually involves the placement of the child with a responsible relative or in a well supervised foster home. This placement may be temporary or permanent depending on the magnitude of the problem concerning the family environment and the degree to which it may be corrected. It is well to remember, that it is usually only one child in a family that is physically abused. One wonders what will happen when the abuser's sole target for aggression is removed from his grasp. Will he turn his attention to yet another young

member of the family? If this be the case, then the problem has not been solved, but instead has been magnified two-fold.

On the other hand, it frequently happens that the non-abusive parent separates from the aggressive spouse in order to protect the battered child. However, a few months later she becomes reconciled with her husband and the child once more is in a situation where he is vulnerable to further injury. It is this all-forgiving naive attitude plus the fact that the continuing physical and emotional danger to the child cannot be seen and verified which plague the social agencies and court attempting to deal with cases of the BCS.

The Nutritional Variant

So far only those children presenting with bony and soft tissue injuries and other manifestations of repeated mechanical trauma arising from commissive acts of physical abuse have been considered. As mentioned previously, the picture of the BCS would not be complete without some mention of the acts of omission whereby the child does not receive adequate nutrition. However, this type of maltreatment is more subtle and covert and therefore more difficult to diagnose and manage.

It is similar to the BCS because there is a complete discrepancy between the facts concerning the child's condition related by the parents and the clinical picture of starvation, dehydration, and marked weight loss; such a clinical picture just couldn't have developed over the short time interval stated by the parents. The chief complaint may be that the child is a poor eater or that he is not doing well (failure to thrive). Unfortunately, many of the examining physicians are too prone to explain the child's cachectic state by a host of diagnostic possibilities which require the expenditure of much effort, time, and money before each one can be excluded, rather than include starvation due to neglect, ignorance, and thoughtlessness in their differential diagnosis.

However, once the diagnosis has been made the appropriate authorities should be contacted in order that an investigation of the home situation can be undertaken immediately in order to prevent recurrences. Ideally the welfare agencies should search out instances of child neglect before his condition deteriorates to that of an irreparable state. To attain this goal is indeed very difficult. Investigation of the home and family background of these malnourished children revealed long periods of willful neglect and callous disregard for the child's plight coupled with unwillingness and failure to seek readily available medical attention. In those cases where death resulted, the infant had not been seen by a doctor or a visiting nurse for at least three or four months prior to his death.

Conclusion

In January 1962, the Medical Records Committee at the Hospital for Sick Children recognized the BCS as a clinical entity and assigned to it the code number 010-400B. Since its introduction, very few case histories of abused or starved children have been recorded under this code. Unfortunately, this does not indicate a low incidence of such cases, but demonstrates the unwillingness of the attending physician to diagnose these as clinically Battered Children. Perhaps over the years to come there will be an actual decrease in the incidence of these cases so that the BCS will eventually become a rare clinical entity.

One can only hope that practicing physicians at the present time and those now in Medical Schools who will be practicing in the future, will be aware that such a syndrome involving purposeful physical abuse or starvation of helpless infants does exist. But awareness is not enough. Whenever he is confronted by a child with suspicious skeletal or other injuries, the conscientious physician should vow to himself that he will do his utmost to explain the etiology of the injuries by a thorough investigation of the child's environment. This should then be followed by appropriate management. In so doing, the physician will find himself in a better position to correctly diagnose and treat cases of the Battered Child Syndrome.

At this time, I would like to extend my heartfelt thanks to M.J.A., G.E.M., and the librarians at the H.S.C. without whose assistance this article would not have been possible.

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(see page 246)

The Juvenile Diabetic

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Diabetes mellitus is best understood as a syndrome characterized by hyperglycemia and glycosuria, associated with abnormal metabolism of fat and protein. Although, technically, a relative lack of insulin may be a good definition, from a practical point of view, the disease is best defined as a loss of the regulation of insulin release. Diabetes mellitus is seen in children as a simpler form than in adults due to the infrequent findings of associated degenerative disturbances in juvenile diabetics.

Incidence

Recent studies show a ratio of 1 diabetic per 2,500 population under 15 years of age. Taking the total diabetic population, the incidence in the age period of childhood is estimated to be from 5-8%. In adults, diabetes mellitus has been shown to be more common in females than in males, but this has not been substantiated in children.

Etiology

The assumption that heredity is an important factor predisposing to diabetes is strongly supported by recent studies. However, the exact mode of inheritance is not clearly defined. One series showed a family history in 62% of a group of juvenile diabetic patients living longer than 15 years. Dr. Chute obtained a positive family history in 43% of a similar group.

Pathologic Physiology

The two major groupings of diabetes are:

1) Growth onset (juvenile) diabetes
2) Adult onset (maturity) diabetes

To understand and to treat the juvenile diabetic, a knowledge of how the pathology and the physiology differs from the adult diabetic is essential. The most striking difference is that in the juvenile diabetic there is an almost total absence of beta cells in the pancreas and these have a minimal or no insulin secretion. The beta cells in the adult type though reduced, are present and the insulin content may be 75-80% of the normal value.

The fundamental cause of the diabetic state is nevertheless identical in both types of diabetes; i.e. the deficiency of insulin secretion by the beta cells of the pancreatic islets. Pathologically, in children, there is characteristically a lymphocytic infiltration of the islets of Langerhans. This is believed to be secondary, however, to a pre-existing injury rather than a primary cause. There is usually a lack of correlation between the severity of the diabetes and the pathological changes.

The alterations in metabolism are identical to those in adult diabetes. Fat and protein are oxidized at excessively high rates when carbohydrate is not available resulting in body protein breakdown and the accumulation of excessive quantities of ketone bodies in the blood. The factors that are to some degree operative in the development of acidosis are:

1) Increased accumulation of ketone bodies in the blood.
2) Loss of fixed base with the renal excretion of acid.
3) Dehydration secondary to the hyperglycemia and acidosis resulting in impaired kidney function.
4) Accumulation of acids which are ordinarily excreted by the kidney.
5) Late cardiovascular decompensation resulting in lactic acid accumulation.

In the non-acidotic state, the characteristic chemical change consists of glycosuria and hyperglycemia with or without ketonemia. The severity of the diabetes bears no direct relationship to the level of the blood sugar. However, the degree of ketonemia is a good guide in patients developing acidosis and coma. At levels of 80-100 mg/100 cc., there is severe acidocis; whereas, at levels of 20-30 mg/100 cc., there may be no associated acid base disturbance. As a general rule, the cholesterol levels and blood lipids are not elevated if the diabetes has been controlled.

How does the maintenance of normal glycemia in a juvenile diabetic differ from the adult diabetic? The following scheme is useful in that it depicts the maintenance of a normal glycemia as being dependent upon a balance between;

carbohydrate intake + stored carbohydrate
+
conversion of other foods to carbohydrate

vs.

basal metabolic need + energy for activity.

This balance in turn is regulated by hormonal factors—insulin, somatotrophic hormone, ACTH, adrenal cor-

tical hormones. But these hormonal factors are probably operatively functional in identical fashion in adults and in children. It has been postulated, therefore, that the real difference in the maintenance of a normal glycemia lies in these other factors such as:

1) Metabolic rate — since this is relative to surface area, this rate is increased in children since they have an increased surface area in relation to their weight.
2) Activity — Children have an increased activity in comparison to adults.
3) Eating habits — Children have a higher intake of soluble carbohydrate.
4) Infections — Childhood is a period of repeated infection until adult level of immunity has been reached.
5) Emotional factors — Children are more emotionally labile.
6) Technique of insulin administration — Children are more prone to errors in technique, a few of which are:
   a) Failure to suspend a precipitated insulin preparation prior to administration.
   b) Frequent intradermal injections rather than sub-cutaneous because of fear and apprehension.
   c) Failure in rotating sites of injection resulting in sclerosing of site of injection with subsequent impaired absorption.

Clinical Manifestations

1) The onset of the disease is much more rapid than in adults. Not infrequently, the presenting symptoms are coma and stupor.
2) The child is underweight rather than obese, as is often the case in adults.
3) Symptoms — wasting, polydipsia, polyphagia, polyuria, bed-wetting in a previously toilet trained child, dry skin, pruritis, skin infections.

The precomatose child presents with drowsiness, dryness of skin, flushed cheeks, acetone breath, hyperpnea, nausea and vomiting, abdominal pain, and often generalized body pain.

Diagnosis

Diabetes mellitus must be considered in the following types of patients:

1) A child who has history suggestive of diabetes.
2) A child with transient or persistent glycosuria.
3) A child who has clinical manifestations of acidosis with or without stupor or coma.
4) A child previously toilet-trained who begins bed-wetting.

The diagnosis in all instances depends on lab data.

Differential diagnosis

Differential diagnosis must include hyperpituitarism, hyperthyroidism, adrenal tumour, acute infections, cerebral injuries, emotional disturbances, lead poisoning, renal glycosuria (uremia, encephalitis and other intracranial lesions must be considered if patient presents in diabetic coma).

Treatment

The aims of diabetic management are:

1) The correction of the underlying metabolic abnormalities by insulin therapy.
2) The attainment and maintenance of ideal body weight.
3) The prevention of complications commonly associated with the disease.

The initial stabilization of the new diabetic and the immediate regulation of blood sugar levels follows a common routine, easily found in up to date therapeutic textbooks. The limited space of this article does not allow its inclusion. The principles of long term treatment, however, will be discussed.

A large number of paediatric clinics have adopted the practice of using the so-called Free Diet on the basis of the claim that complications are an inherent feature of the diabetic disease. Frederick Allan shows, however, in his study that complications diminish in proportion as the control is thorough and that complications vary with the degree of control. If the latter school of thought is accepted, the principles of long term treatment are as follows:

1) *Insulin*
   a) Oral insulin substitutes are contraindicated in the treatment of the younger diabetic patient.
   b) As younger diabetics grow older, their diabetes becomes more stable allowing discontinuance of insulin doses other than daily morning dose.
   c) Appropriate dose alteration must be made for the inexplicable slow rises and falls which take place in the juvenile diabetic.
   d) Where unusual amounts of physical exercise are anticipated, the dose of morning injection can be increased or extra nourishment can be ingested immediately prior to the exertion.

e) Hypoglycemic reactions are to be avoided at all costs. Patients should carry with them at all times glucose containing hard candies and patients and relatives should be familiar with indications for other methods of glucose administration.

f) As patient grows and caloric requirements increase appropriate increases in insulin dosage must be anticipated.

g) Occasional days of unusual inactivity or the intervention of mild febrile illness may require accessory doses of regular insulin according to the routine sliding scale.

h) Daily urine testing before breakfast and before supper is mandatory and should be enforced.

2) *Diet*

a) Progression up the caloric scale of A.D.A. diets as child grows, should be followed with concomitant increases in insulin dosage.

b) As the young diabetic progresses socially, he should be guided as to the intelligent selection of refreshments, snacks, etc.

3) *Additional Measures*

a) Supplementary vitamins may be given.

b) Consistent patient support, reassurance and encouragement by the physician and parents are essential in periods of depressions and rebellion.

c) Continuous emphasis by the physician on the fact that their choice of vocation, marriage, athletics etc. are not impaired by their disease.

d) At least, semi-annual complete physical examinations by their physician are mandatory.

e) Recognition and treatment of serious psychologic difficulties which not infrequently arise: states of anxiety over the disease and its complications, false implications of invalidism, rebellion against regimentation, jealousy of their nondiabetic siblings and playmates.

The management of diabetic coma should follow the routine regimen and should include insulin, intravenous fluids, electrolytes, and additional measures as warranted. Similarly, the establishment of maintenance therapy of the non comatose new diabetic should follow the well outlined routine regimen with particular emphasis on insulin and diet.

Late Complications

The most serious problem now faced by the juvenile diabetic is the prospect of vascular degenerative changes in adult years. Regardless of the age of onset in different patients, hypertension, albuminuria, nephropathy, hemorrhagic retinopathy and cataracts appear with increasing frequency in the second and third decade of the disease, and may be found in 90 to 100% of all cases toward the end of the third decade of the disease. In one series among patients who had their disease longer than 10 years, 14% had perceptible changes of the lens, 7% had clinically significant cataracts, 43% had diabetic retinit's, 16% had hypertension and 4% had albuminimia.

Most observers agree that the late complications are directly correlated with the duration of the disease and that repeated attacks of ketosis and acidosis constitute an obvious hazard.

Course and Prognosis

The life expectancy of a child with diabetes mellitus before the introduction of insulin in 1922 was 2 years. In a series done by Dr. Chute on patients with insulin treatment, he reports the survival rate around 50% in a group of 123 patients where diabetes mellitus had started during childhood and had existed for 15 or more years. All of his patients (24) who had survived 20 or more years had serious degenerative lesions. The preliminary work of Fajans and Conn has shown a faint hope for the prevention of diabetes mellitus if the trend is discovered early. By the administration of tolbutamide to children with only a moderate elevation of blood sugar but a diabetic blood sugar curve, they have shown a complete return to normal which suggests that the production of insulin by the residual beta cells of the pancreas is enhanced and stabilized by such treatment.

ACKNOWLEDGMENT

I wish to thank Dr. A. L. Chute for his direction and constructive criticism of this article.

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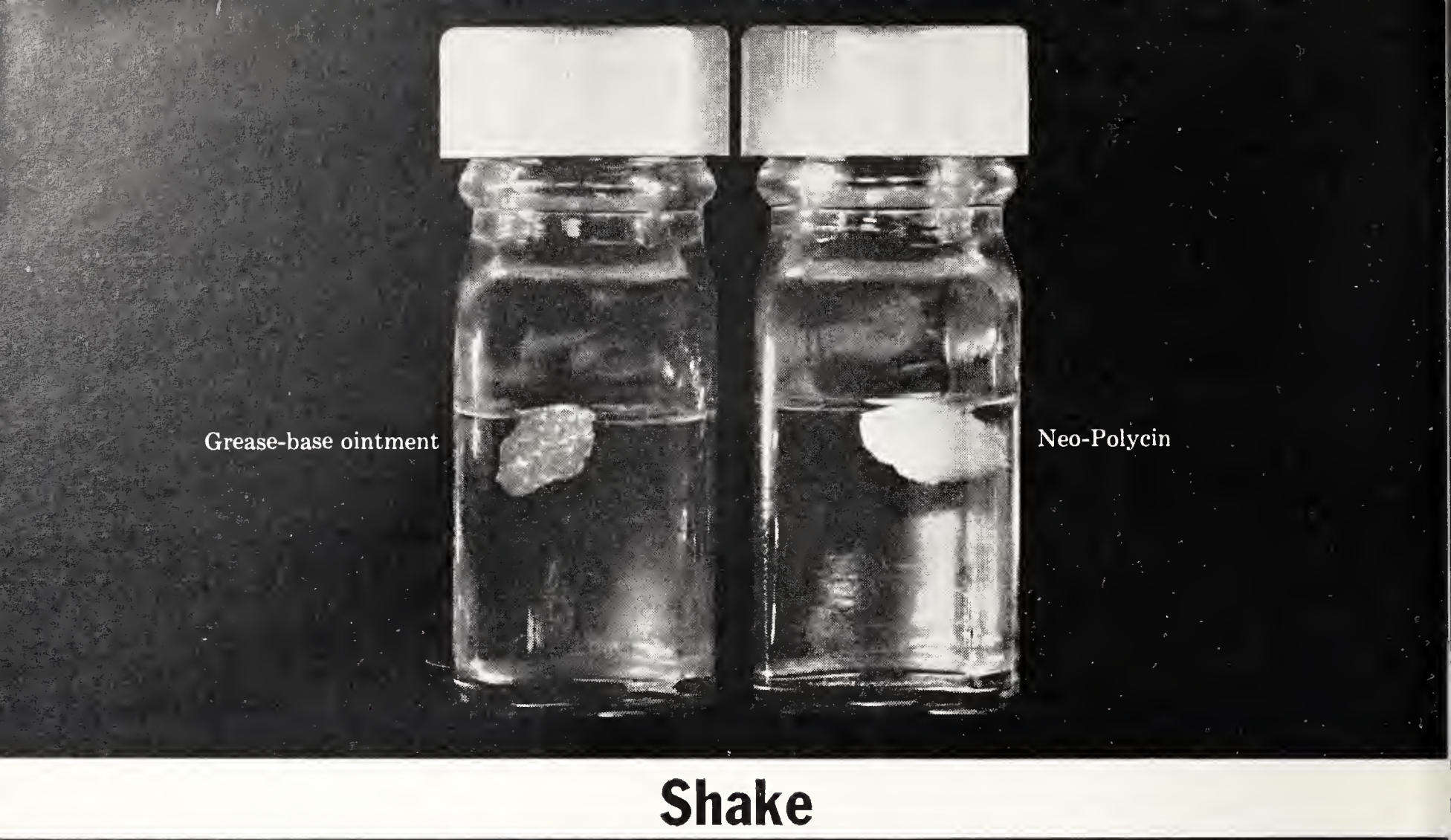

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Juvenile Rheumatoid Disease

Mel Petersiel 6T5

"There is a disease, occurring in children . . . characterized by elastic fusiform enlargement of joints . . . by (an) enlargement of glands and spleen . . . (which) is not shared by the rheumatoid arthritis of adults . . . There (also) occurs in children a condition which appears to be identical in every respect with rheumatoid arthritis"[1]

Few authors since George Frederick Still wrote this in 1897 have cared to make his careful distinctions. The British investigators, with the greatest experience in the field, term any rheumatoid disease arising in children — "Still's disease". Others would reserve the eponym for a more florid disease, replete with pericarditis, splenomegaly and hepatomegaly. This latter affection, involving 15-20% of children with rheumatoid arthritis, would appear closer to Still's original description. But, differentiation is hazy, for the rheumatoid disease of children does not present in the adult manner.

In children a severe systemic disease is usually seen, involving different joints, in different ways, having fewer positive serological tests, being of more acute onset, and having a definite intermittent course. Yet many of the findings are also present in the adult disease, and in both types of childhood disease. We seem to be left with a mysterious entity bereft of eponymic and descriptive protection.

It has been argued that juvenile rheumatoid arthritis is a generalized, systemic disorder, and should more correctly be termed juvenile rheumatoid disease. The systemic manifestations would then allow classification under the "collagen" diseases, where the disorder belongs. The same shift of emphasis appears to be occurring in the adult disease, depriving us again of any singularity in the juvenile. The demonstration of similar incidences of positive rheumatoid factor tests in relatives of patients with juvenile and adult rheumatoid disease tends to reinforce the connection.

Etiology:

As is customary in a collagen disease, infections, emotion, and extremes of temperature have all been implicated as etiologic agents. While they may appear to precipitate attacks, the more popular area of autoimmunology has been invoked as the major culprit. It is believed that an incomplete macroglobulin — which behaves as a degraded antibody — causes rheumatoid arthritis in a patient capable of recognizing this as a foreign protein. A great deal of research is presently attempting to clarify the picture.

An interesting sidelight is that common histological and serological findings have been studied in systemic lupus erythematosus, Hashimoto's disease, and rheumatoid arthritis, again increasing the evidence for an autoimmune origin.

Heredity:

Most studies of genetic patterns reveal a greater incidence of spondylitis in male relatives of these patients, and strangely enough, of erosive arthritis in females. Bywaters believes this indicates a double linked inheritance, of spondylitis and "sero-negative" rheumatoid disease — juvenile disease without a positive Rheumatoid factor test.

Incidence:

Two point seven of 100,000 children under 15 are affected, a number similar to that of juvenile diabetes and the nephrotic syndrome in children. A varying age of incidence depending on the type of disease is also seen. The infantile, systemic type occurs between the ages of 1-3, with males and females involved in equal numbers. The more adult type occurs around 5 years of age, with females outnumbering males 2 to 1. A second peak of incidence is noted before puberty.

Clinical Manifestations:

Myriad methods of onset, from pain and swelling in a single joint to generalized or local systemic disease, confuse the picture, but the more typical aspects will be described.

General: The disease may appear with malaise and irritability. In later stages, there is obvious apprehension with guarding of joints; periarticular swellings are seen.

1. Fever: the most common sign demonstrates either unremitting or shows a diurnal variation. This may be characteristically elevated in the evening (to 105°C) and normal during the day. In 60% of cases, fever appears before joint involvement often leading to a diagnosis of pyrexia of unknown origin (PUO).
2. Rash: Often the most characteristic sign of the disease, it appears in from 30-75% of

patients, and is customarily seen only when the fever is high. It is described as an almost pathognomonic fleeting, evanescent, salmon-pink, discrete maculopapular rash, non urticarial, and occurring over the entire body. The eruptions are erythematous and circular, may involve the face, and do not spread. (The latter two characteristics are typical of the erythema marginatum of rheumatic fever).

3. Lymphadenopathy is generalized, and is an early manifestation. It is seen in 10-50% of cases.

4. Splenomegaly is often termed part of "Still's" disease, rather than of juvenile rheumatoid arthritis — but again only by those who feel there is a distinction. This point will be discussed under "prognosis".

5. Heart involvement is of three types. Pericarditis is most common, with typical friction rub findings. Myocarditis is rare, as is aortitis.

6. Eye involvement: A specific type of affection, band keratitis, is commonly seen. It may precede, as a single sign, any other involvement for up to 2½ years. An iridocyclitis may also be present.

7. Subcutaneous Nodules: These are considered to be similar to the nodules of rheumatic fever, and occur much more rarely. Their presence is often associated with a positive rheumatoid factor test.

8. Joint manifestations: Large joints are characteristically involved first. One half of the cases are monoarticular at onset, and up to 10% will remain so throughout the course of the disease. The most commonly involved joint is the knee, followed by wrist, ankles, carpi, hips, tarsi, hands and cervical spine. The cervical involvement varies from that of the adult.

   First signs are commonly arthralgia, with no inflammatory signs, and morning inactivity, similar to the morning stiffness of the adult disease.

   Hand involvement results in fusiform swellings of the PIP joint, redness, heat and tenderness. The skin overlying the spindle shaped fingers becomes smooth and shiny. DIP, MP joints may also be involved.

Xray findings:

Thesc usually appear late in the disease course. Originally there are soft tissue swellings, with osteoporosis and growth changes. Subperiosteal new bone may be seen. Bone erosions, and periarticular bone cysts are common.

Sacroiliac changes are noted, as are cervical spine manifestations. Bony ankylosis is seen on these areas, leading to a loss of cervical lordosis, and growth defects.

Growth Defects:

Some of the most serious findings in this disease are changes in growth due to joint involvement. Destruction of the epiphysis, epiphyseal plate by disease action results in local shortening, hastened by fibrous, then bony ankylosis. Changes are seen in fingers, in the growth of the jaw when the temporo-mandibular joint is involved, and in the spine, leading to reduced stature.

Diagnosis:

The customary rigid diagnostic criteria of adult rheumatoid arthritis do not appear to be applicable to children. More commonly such qualifications as polyarthritis of 4 joints, present for 3 months, are significant. Synovial biopsy for the characteristic pathological changes is often used in diagnosis. The classic finding is a villous, hypertrophic synovitis, with intensive fibrin deposits on the synovial surface. Pseudofollicles — aggregations of lymphocytes — are often seen. Palisading of fibrocytes in the region is common.

Laboratory aids:

The blood picture is that of a normochromic normocytic anemia, often with a leukocytosis of up to 30,000. In the active phase, the nonspecific ESR (done by the Wertergren method) is raised. There is a mild rise in gamma, alpha-two globulin.

Tests for the adult disease are based on the presence of the Rheumatoid Factor, an altered globulin. This is rarely found in juvenile disease, and the common Rose Waller, latex and bentonite fixation tests are positive in less than 15% of cases. More sophisticated inhibition tests are positive in some series in up to 87% of cases, but these techniques are not commonly available. No simple diagnostic test has yet been developed.

Differential Diagnosis:

Three areas require varying differentiation, and are presented in tabular form.

A. Systemic Manifestations:

I Acute Infection

1. poliomyelitis — muscle stiffness, GI symptoms, pharyngitis
2. encephalitis — meningeal irritation, lethargy.
3. septicaemia — infective focus.

II Collagen diseases

1. Systemic lupus erythematosus — scaly, polymorphous rash with petechiae, LE cells, renal involvement.
2. Rheumatic fever — discussed below
3. Other collagen diseases — rare in childhood — polyartertis, dermatomyositis, scleroderma have characteristic skin and/or muscle involvement.

B. Joint Manifestations:

I Monarticular

1. Post-traumatic
2. Tuberculosis — synovial fluid smear, culture, synovial tissue biopsy, Mantoux test.

II Polyarticular

1. Rheumatic fever — associated with pancarditis, pansystolic and diastolic murmurs, conduction defects on ECG, variable fever pattern, flitting joint involvement, high ASO titre, higher ESR, preceding strep. infection, lack of joint destruction.
2. Polyarthritides of ulcerative colitis, psoriasis, Whipple's disease, and very rarely in children, ankylosing spondylitis.

C. Growth Disorders:

1. Arthrogryposis congenita
2. Morquio's disease

Course and Prognosis.

Most reasons for differentiating juvenile rheumatoid and Still's disease are centred here. The more vivid manifestations of Still's disease are mirrored by its more serious prognosis, with serious joint involvement, and eventual typical adult disease, with joint destruction, subluxation, and ankylosis. Juvenile rhuematoid disease, on the other hand, customarily runs a relapsing, remittent course, often with complete cure before puberty.

Acute cases may die of the disease, untreated, in 6 months. Chronic joint residua will result in other untreated patients. If seen early and treated, 50% of all patients will recover completely, 25% will have only moderate disability, and a further 25% will be severely crippled. Amyloidosis, pathological fractures, vertebrae collapse may occur in severe cases. Surgical treatment, to allow more normal joint action, may be called for with permanent joint changes.

Treatment:

It can readily be recognized that this is a severe, chronic disease, requiring major efforts in treatment. Not only is it a severely disabling disease but it is also a serious psychological burden, both for the child and for his family. To attack on both physical and psychological fronts involves almost every facet of medical, surgical and supportive therapy. The major factors involved are these:

1. Rest: Bed rest in the acute condition is imperative. Throughout other periods, sufficient daily rest must be alternated with exercise and activity.
2. Heat: This will prove a major help in relief of pain and stiffness, allowing more joint mobility and focusing less attention on symptoms.
3. Splinting: In the acute phase, splinting involved joints will allay pain and anxiety. Over a longer period of time the use of splints will prevent much of the deformity associated. Night splinting of small joints, neck collars are often used.
4. Exercise: Retraining of disused, painful muscles and joints must be begun professionally. The importance of graded exercises in physical rehabilitation cannot be overemphasized, for only with them can the patient realize his capabilities. Home programs are available after initial treatment.
5. Drugs: Probably the least important aspect of therapy, drugs are usually used in the following order.

   a) Salicylates — kept at a high level in the blood by frequent dosage. Toxic signs are well known, and must be watched for. By

relief of pain, muscle spasm is prevented, easing the condition.

b) Steroids — due to the customary short-term character of the disease, steroid therapy is more popular in this condition, with less reticence about side effects. Cortisone or prednisone is used, especially with serious systemic involvement.

c) Phenylbutazone, antimalarials, and gold have found less use in childhood disease than in the adult.

6. Most important of all measures is social rehabilitation. The facts about this disease must be made known to parents and to patients. There must be encouragement at all levels, both to understand capabilities and limitations, and to guide through the years of chronic disease. Normal activities, school attendance, association with normal playmates, and a lack of favouritism and overattention will have much to do with the outcome of the disease. Understanding, sympathetic care will allow normal personal adjustment and development of the child, but psychiatric care will often be required if this fails. Financial and psychological aid must be available to the parents, for this is a long term, mystifying and saddening disease.

The cure of a chronic disease state, especially in a child, is a gratifying thing for a physician. Every bit of his professional and personal skill, and an infinite patience are required. But the end result is worthwhile, for a failure in this disease will result in both an emotional and physical cripple.

ACKNOWLEDGEMENT

I would like to thank Dr. P. Rosen, of the Mount Sinai Hospital for time and aid in the preparation of this article and Dr. T. Yates of the Hospital for Sick Children for kindly providing X-ray photographs.

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(cont. from page 230)

The importance of the recognition of perceptual disturbances in a child lies mainly in the fact that these children are often amenable to therapy. Just as many of us can improve our ability in some sport by continual practice, so these children will frequently improve if their input-output mechanisms are properly managed. Similar to the "critical periods" occurring in the development of animals during which specific forms of behavior are learned and after which the behavior learned cannot be unlearned, so there are similar periods in humans. However, the optimum period lasts longer in humans than in animals, and if a child is delayed in any phase of development, he may still catch up, especially with skilled help. Perceptual development normally occurs between 3 and 7½ years, and a child with perceptual difficulties who receives perceptual training at this time has a good chance of overcoming his difficulties, particularly if training is commenced early in this period.

Thus, as medicine delves deeper into the fields of psychology and psychiatry, more and more facets of the human psyche are being revealed. While numerous new questions are being posed, at least a few of the old ones can now be answered.

BEK

I would like to thank Dr. W. A. Hawke for his assistance in preparing this article.

Acne Vulgaris — A Teenage Problem

Edward G. Davies, 6T4

Every thoughtful physician appreciates the problems that Acne Vulgaris presents to teenagers. This disease with its scarring and unsightly lesions on exposed parts of the body can be sufficiently serious to affect the psyche, even to the point of producing deepseated and permanent feelings of inferiority and insecurity. Moreover, acne in a child, will often affect the entire family, initiating misunderstandings, reproaches, tensions and lasting unhappiness. This burden borne by the adolescent, is eased somewhat by the fact that acne vulgaris is so common, affecting from 60 to 90 per cent of young men and women between 12 and 20 years of age.

Acne Vulgaris in the adolescent may be referred to as a 'physiologic' disease in the sense that it occurs at the very time that the hormonal system undergoes maturation as a normal physiologic process. Thus, it will be found that ordinarily the disease has its onset shortly before or coincidental with the first signs of puberty. The most common offenders among foods and drugs taken by the adolescent include: chocolate in any form, sea foods, nuts, cheeses, iodides and bromides. Further, the history will frequently reveal the fact that the individual's acne became distinctly worse a few days after cessation of sun exposures. There are also those patients whose acne, particularly that of the forehead and temporal areas, becomes unmistakably worse in association with seborrhea of the scalp. Today there are few patients who do not report the fact that an emotional stimulus of one type or another contributed to a flare-up of their acne. In some cases, the emotional disequilibrium itself seems to be sufficient to trigger the exacerbation. While nothing can be done as yet about hereditary factors which may play a role in the patient's disease, it is often worthwhile to ascertain whether the parents had acne. If there is a history of acne, it is wise to know what type and the degree of severity. More important, the physician should not lose sight of those external factors which aggravate this disease, such as greases, waxes and oils, cosmetics and certain clothing.

Pathogenesis: The organ of response for the acne lesion is the pilosebaceous apparatus which consists of a hair follicle and its sebaceous gland. The acne lesion is the result of partial or complete obstruction of this gland. This obstruction is caused by increased formation of an altered form of sebum. In addition, there is proliferation of the epidermal cells around the opening of the sebaceous duct. These changes are precipitated in an imperfectly understood manner by sex hormones. The result of these changes is that there is an inspissation of sebum which swells the duct and forms what is known as a comedo or blackhead. This blocks the flow of fluid, which may be infested, from the surface, forcing it laterally and deep. Thus, pustules and cysts are created. Sometimes, the comedo may develop to such a size as to leave behind it atrophic scarring due to pressure. Where there have been repeated attacks of suppuration the affected areas are likely to be deeply pitted, and ugly nodular thickenings are often left in the texture of the skin. While the area most commonly involved by acne is the face, similar involvement may affect the shoulders, upper back and the chest. In addition there is often oiliness of the face, particularly the forehead, glabella, nose and the paranasal zones. It is here that the skin may appear thickened and the orifices of the pilosebaceous units enlarged because of loss of hair.

Treatment:

1. General: One should always correct any intercurrent affection. Teeth should be examined for caries. Appropriate treatment should be prescribed for any seborrhoea in the scalp. Investigate the state of the digestion, and in women, of the menstrual functions. The diet should be such as the patient can digest easily. Greasy foods and highly spiced, made-up dishes should be avoided.

2. Local Treatment: (A) Removal and prevention of the follicular plugs. The foundation principle of topical acne medication is the achievement of a continuous mild nondisfiguring drying and peeling of the skin to remove and prevent follicular obstruction. For the mild case the thorough use of soap and water may be sufficient. Special soaps are available. Ordinarily, washing and cleansing alone are not enough to relieve the papular and papulopustular types of acne and the use of drying preparations is required. These are preparations containing resorcin and precipitated sulphur which may be rubbed into the skin in order to produce mild dryness and peeling. It is always wise to begin with weaker concentrations of the drying and peeling agents, increasing the strength only when they fail to produce the desired response after one week's use. At times excessive and irritating dryness and subsequent peeling will result from some of these preparations. When this happens discontinue temporarily and use instead a soothing and if necessary antibiotic ointment or lotion.

Removal of the Comedo: It is frequently necessary to remove them by mechanical means. Many comedo extractors have been devised, the simplest is the best, and this is a tapering glass tube with thick edges. After washing the face with soap and water the narrow end of the comedo extractor should be applied over the blackhead, and steady pressure applied until it is seen to issue from the duct into the bore of the tube.

(B) Relief of Infections and Inflammation in and around the Pilosebaceous unit: Compresses — When the pustular lesions and cysts are numerous, hot compresses are usually of marked benefit. These may be either simple ones such as normal saline or Burow's solution. These compresses should be applied for 20 minutes twice daily.

Antibiotics And Sulphonamides — Use systemic antibiotics such as Tetracycline Hydrochloride in combination with Nystatin for managing the pustular and cystic forms of acne. Every effort is to be made to reduce the daily dose to the smallest effective level. This is around 125 mg every other day, in cases on long term therapy. Among the sulphonamides use Sulfisoxazole· beginning with 0.5 grams 4 times daily for 1 week and, if well tolerated and necessary, increasing the dose to 2 grams 4 times daily.

Hormones — Despite the evidence that common adolescent acne is undoubtedly in the main based on hormonal effects, modern hormonal therapy has not yet proved of more than occasional or partial value in its management. If the patient is over 18 years of age, and other measures have proved ineffective or insufficient, endocrine therapy should be given a trial. This is particularly so in persistent acnes of females and especially when exacerbations are proved to be chronologically related to menses; and in the severely cystic or congloberate type of acne whether related to menses or not, and whether in males or females.

Vitamin A — The value of the oral administration of Vitamin A in the management of acne vulgaris is controversial. It can be of value however in some cases in influencing the keratinization of the epidermis in the comedone and papular types of acne.

External Therapy which can be administered by a specialist:

1. Ultraviolet Light — This is used to produce superficial peeling of the involved areas of skin. It is not unusual, however to see a mild flare-up in the acne when these lamps are used sporadically. The treatment is effective when used in carefully controlled amounts.

2. Cryotherapy — Solid carbon dioxide is often beneficial in hastening the resolution of some of the individual papular and papulopustular lesions. For generalized peeling of the face, carbon dioxide slush produces immediate redness. This procedure has now been largely replaced by wire brush planing or dermabrasion.

3. X-Radiation — Properly administered and in the correct doses, this remains a very effective means of treating cases of acne which are stubborn in their response to the other forms of treatment.

4. Dermabrasion — In selected cases of acne scarring the use of this procedure is a distinct aid. In the cases finally chosen for planing, the procedure has proven safe and has achieved an estimated 30 to 90 per cent improvement in the patient's appearance.

Conclusion: The patient with acne must be made aware of the fact that the treatment must be continued regularly for the entire period of adolescence or longer. Acne is still one of the most distressing of all human ills. Fortunately, it is also one of those most benefited by well chosen, properly employed, modern therapy.

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CORRECTION

It has been brought to our attention by Dr. J. S. Prichard that point #5 on page 60 of the second issue should read — "seizure lasting less than thirty minutes" instead of thirty seconds as printed.

Two Hip Conditions Common in Childhood

Ronald B. Filderman, 6T4

IDIOPATHIC MONARTICULAR SYNOVITIS

Incidence

Idiopathic monarticular synovitis is said to be the most common cause of transient pain and spasm in the hip during childhood. It is found in the 3 to 10 year age group, with 80% of the cases occurring in males.

Etiology

As the name of the condition implies, the etiology is unknown. There is often a respiratory illness, usually a streptococcal infection, 10 days to 3 weeks preceding the onset of symptoms. The condition may therefore represent an allergic manifestation in the joint. Occasionally, there is a history of recent joint trauma.

Clinical Features

The symptoms may be quite severe, mimicking those of a septic hip joint, or, on the other hand, so mild that they go almost unnoticed. In most cases, pain is experienced in the knee during the first 24 hours, and thereafter, gradually localizes to the hip. The child who experiences moderately severe pain, prefers to limp or to stay off his feet entirely, because the pain is then diminished. The hip joint may be slightly tender to anterior and posterior palpation. Limited abduction and internal rotation along with temporary flexion contracture always serve to somewhat restrict active movement. Passive movement is limited by muscle spasm. The child usually has a temperature of about 101°F., a moderately elevated sedimentation rate, and a normal or slightly elevated white blood count.

Radiographic Changes

On an anteroposterior view of the hip joint, there is swelling of the capsular shadows, best seen superiorly and laterally, and a widening of the joint space, best seen inferomedially. There are no bony changes.

Differential Diagnosis

Septic involvement of the hip, slipped epiphysis, congenital dislocation of the hip, tuberculosis of the hip, rheumatic fever or rheumatoid arthritis, and Legg-Perthes' Disease must all be ruled out before the diagnosis of idiopathic monarticular synovitis is made.

Treatment

Continuous traction in line with the flexion contracture should relieve the symptoms rapidly. When the child does not bear weight, the inflammation about the hip tends to diminish readily, giving rise to immediate pain relief. It usually takes from 36 to 72 hours to relieve the muscle spasm. An additional 1 to 3 weeks of non weight-bearing is advisable after this, during which time full hip motion is regained.

Prognosis

If the spasm has not been relieved in 4 to 5 days, a more serious underlying etiology should be considered. During the ensuing 6 months, approximately 5% of patients with synovitis of the hip develop the radiographic changes of Legg-Perthes' Disease, and any recurrences of the 'synovitis' should bring to mind this possibility.

LEGG-PERTHES' DISEASE

A large group of childhood conditions exist in which the basic lesion is an idiopathic vascular disturbance of an ossification center. The commonest and most serious of these conditions (the Osteochondroses), occurs in the capital femoral epiphysis where it is known as Legg-Perthes' Disease (Perthes Disease, pseudo coxalgia, Legg-Calve-Perthes' Disease, osteochondritis deformans coxae juvenilis, coxa plana, quiet hip disease).

Incidence

This condition is found in children between the ages of 3 and 12 years, with 80% of the cases in males. Ten per cent of cases occur bilaterally. Negro children are relatively immune to this disease.

Etiology

The blood supply to the femoral capital epiphysis normally arrives via the lateral epiphyseal branch of the medial femoral circumflex artery, and via the medial epiphyseal branch from the obturator artery (through the ligamentum teres). These vessels anastomose and send branches toward the articular cartilage.

In this disease there is an interference with this blood supply, leading to ischaemia and avascular necrosis of the ossification center. The precipitating factor remains

unknown in most cases. Many theories of causation exist, some of which are trauma (tearing or thrombosing of vessels), congenital and hereditary factors (common after congenital dislocation), inflammation (synovitis or infection), embolism, and thyroid dysfunction.

Clinical Features

The early clinical features are variable. There is usually a history of limping for several weeks (possibly only intermittent), together with pain at the knee, often referred to the inner thigh or groin. The pain varies in intensity, being increased with activity and relieved by rest. There is almost invariably some limitation of hip movement, particularly in internal rotation, flexion, and abduction. The hip joint may be tender to anterior and posterior palpation. Atrophic thigh muscles may be found on the affected side. In some cases, the symptoms may be so mild that only careful examination of hip movement will lead to the diagnosis. Occasionally, abdominal X-rays, taken for various reasons, reveal the changes of well advanced but asymptomatic Legg-Perthes' Disease. The characteristic limitation of motion can usually be demonstrated in these children. When the disease presents bilaterally, there may be a waddling type of gait and a positive Trendelenburg test.

Pathologic And Radiologic Changes

The disease process spans a 2 to 6 year period, divisible into 3 active stages and a fourth or healed stage.

First Stage

This stage lasts from 2 to 8 weeks. After blood supply deprivation, bony alterations take longer to appear than soft tissue changes. Mild inflammation of the synovia and joint capsule leads to swelling and subsequent fibrosis. Radiographically there can be seen swelling of the capsular shadows, minimal widening of the joint space, slightly increased opacity of the epiphyseal center (demineralization of surrounding bony structures which have a blood supply), and an irregular widening of the radiolucent epiphyseal line (often with cystic changes).

Second Stage

This stage lasts from 6 months to 2 years. The articular cartilage obtains its nourishment from the joint fluid and usually manages to survive intact until its bony support collapses. Its surface then becomes irregular, flattened, and spread out. Loss of blood supply leads to the death of a portion of the ossification center. In the surrounding living bone, a reactive hyperaemia occurs and persisting mesenchymal cells generate granulation tissue. Partial demineralization can now take place, and collapse and condensation of the femoral head may occur from stress pressures. Radiographically, increased density of the femoral head, fragmentation (irregular replacement of avascular bone with new uncalcified vascular tissue), and flattening of the epiphyseal center are seen.

Third Stage

This stage lasts from 1 to 4 years and is characterized by the replacement of dead tissue with new bone (or densely collagenized fibrous tissue in which bone slowly forms), and cartilage. Radiographically, the outline of the epiphyseal center gradually becomes regular and intact again, with all radiolucent areas eventually disappearing.

Fourth Stage

The final deformity of the hip joint is characterized by a short, broad, anteriorly-angled femoral neck and an enlarged acetabulum corresponding roughly in shape to the broad, flattened femoral head.

Differential Diagnosis

Idiopathic monarticular synovitis, congenital dislocation of the hip, slipped epiphysis, septic hip joint, tuberculosis of the hip, fracture dislocation, cretinism, Morquio's Disease, Gaucher's Disease, and Hurler's Syndrome must all be differentiated from Legg-Perthes' Disease.

Treatment

With reasonably early diagnosis modern treatment is entirely conservative. It is aimed at restoring full hip motion and protecting the femoral head from forces apt to cause flattening and widening.

In the majority of cases, some form of non weight-bearing is essential in order to prevent the deforming pathological events previously mentioned. A few weeks of continuous traction serve to relieve any muscle spasm and to allow a full range of hip movement to be regained. Thereafter, weight bearing can be prevented by complete bed rest, by utilizing crutches and a belt or sling to hold the knee in flexion, or most commonly, by employing an ambulatory Taylor caliper with a high shoe on the normal side. When the disease occurs bilaterally, the second hip usually becomes involved from 6 months to 2 years after the first. Bed rest or a wheelchair can be used until the first hip has

(see page 253)

Maturational Lag

K. Whytock, 6T4

For years, it has been recognized that some children apparently normal in every other respect, are "slow starters" — that is, they may not speak till much later than other children though they can walk and play the same way. Other children, who seem bright and intelligent, have great difficulty learning to read and write. They have been described under various names — congenital word deafness or the more erudite congenital auditory imperception, congenital aphasia, word blindness, et cetera.

Though the syndromes were well-documented, few workers recognized the connections between them. During the past few years, in our education-oriented society, more attention has been given to these learning disorders and the natural history investigated. Ingram feels that these difficulties represent a form of "Maturational Lag" from which the child makes a complete recovery;[1] others in the field are not as hopeful about the prognosis, pointing to residual difficulties in communication as the probable outcome.

Behaviour is subdivided into 4 main groups — motor, language, adaptive, and personal-social. Maturation in each of these areas proceeds at its own rate and depends on the development of nerve pathways, synapses and biochemical processes. It may:

1) be arrested completely by cerebral damage.

2) be arrested temporarily and never "catch up" to normal.

3) be arrested temporarily but reach normal levels within a few years. This last is referred to as "maturational lag" or "delay".

Though this delay may affect any part of the nervous system it would seem to be most common and most evident in those areas concerned with communication. Maturational lag in this area results in "specific developmental dysphasia (speech), dyslexia (reading), and dysgraphia (writing). These separate but closely allied difficulties of language function are often found together in the same patient or in members of the same family. The family doctor or pediatrician must be prepared to recognize this syndrome and counsel the parents, advising treatment where necessary.

Dysphasia

The presenting clinical symptom is retarded speech development without a proportionate delay in motor, social and adaptive mechanisms. Language development is considered to be retarded if there are no consistently intelligible words by 24 months nor phrases by 36 months; it may be suspected with no words by 18 months nor phrases by 30.

The minute details of the problem are important only to the speech therapist but some of the more gross difficulties are interesting. The severity varies from a simple immaturity of articulation to complete expressive aphasia. Though speech acquisition is slow, the vowels and consonants are acquired in approximately the same order as in normal children. There is an inconstency of defective pronunciation, depending on the position of the consonant in the word or sentence. For example, he may be able to say *"this"* but not *"path"* or *"sun"* and not *"post"*. Abnormalities of articulation are much less evident in imitated speech (as when being coached by worried parents) than in spontaneous chatter (with siblings or friends). Difficulties extend to the area of words and sentences — small words such as "to" or "for" are often omitted or used in place of each other. Syllables in words may be rearranged and syntax poor.

Delay in the acquisition of language may also occur. The child has a limited and immature vocabulary — his speech resembling that of a child 2 to 3 years younger. There may be an associated delay in the comprehension of speech — often, even simple phrases need to be repeated. The intelligent child usually compensates and hides this defect by lip-reading and interpretation of gesture and expression.

Awareness of these speech problems varies from patient to patient, depending as much on the parents' attitudes as on his own insight. Excitement and emotional stress greatly aggravate this condition.

Dyslexia

The basic defect would seem to be in the organization of spatial perception, resulting in a confusion of right and left.[2] This becomes of greatest import at school age when the child begins to read and write. It may be manifest prior to this as difficulty in distinguishing his right side from his left and an inability to tell time — he can't tell which direction the hands are moving — whether the big hand is pointing "to" or "past" the hour![2]

There is an impaired ability to recognize written symbols, to orient them correctly in direction and order and to perceive the significance of their relationships to one another. They often mistake letters for their mirror images and tend to reverse the order of letters in short words and syllables in long. Interestingly enough, it has been found that these children are much better at mirror reading than normals.[3] They may read whole lines backwards — there is a tendency to read one line from left to right and the next from right to left. As in speech difficulties, the intelligent child may more readily mask his problems by memorizing long passages which he identifies by drawings or other marks on the page.

Dysgraphia

Problems in writing are similar to those in reading — reversal of symbols — "b" for "d" (often overcome by substituting the capital), 693 for 396; words — "saw" for "was"; syllables — "tarnish" written "tarshin". In less severe cases, a child is able to write correctly but is too slow for school standards.

Compound Difficulties

Dysphasia, dyslexia and dysgraphia may occur in the same child in various degrees and combinations, and each may be combined with loss of the ability to transmit or appreciate ideas by language in any of its forms. He may have difficulty in breaking words into syllables and finding the written equivalents of spoken words. He may be unable to comprehend the significance of the written or spoken word though he can read and write it correctly.

Diagnosis

Once it has been established that the child's level of performance is below normal, a reason must be found. The first step is to distinguish dysphasia, dyslexia and dysgraphia, irrespective of their etiology, from other causes of slow speech development and retarded learning of reading and writing. Investigation attempts to exclude:

a) local pathology of the articulatory mechanism.

b) deafness.

c) blindness.

d) emotional deprivation.

e) lack of vocal stimulation.

f) severe psychiatric disorders.

g) mental retardation.

In regard to the above, 2 points might be mentioned. First, because of the impairment of speech and writing, it may be difficult to evaluate the I.Q. by conventional testing. However, the Wechler test with its equal weighting of verbal and performance scores and specific sub-tests such as block design and object assembly may be very helpful, not only in determining an estimate of the I.Q. but also in noting specific difficulties such as in spatial perception which may assist in diagnosis.

Second, one must beware, in psychiatric examination, of confusing primary psychiatric disorder with the psychiatric symptoms such as withdrawal, temper tantrums, which may be secondary to speech and educational difficulties. In this respect the history, with emphasis on the chronological development of milestones and of psychiatric symptoms, is very important.

Difficulty may be encountered in differentiating maturational lag from childhood schizophrenia. Here too, history and other symptoms are important. The withdrawn and non-affectionate autistic child has usually been so from birth. The product of a schizoid parent, he has had little opporunity to develop normal relationships and has no need for speech.

In deciding on the diagnosis, one must assess not only the whole child but also his parents and home environment. One needn't be an experienced psychiatrist to form a fairly accurate judgement of the relationships between the child and his parents and siblings.

Having ruled out all of the above, one looks for evidence of cerebral pathology which may account for the disturbance. A complete neurological examination and EEG are usually necessary. In the absence of any demonstrable abnormality, the condition is designated as a specific developmental disorder.

Etiology

While the etiology remains a subject of controversy, certain features of developmental delay are well documented. It is hereditary, probably as an autosomal Mendelian dominant trait with partial penetrance.[4] It shows a predilection for the male — around 3:1.

Developmental delay may occur in a child of any I.Q. or socio-economic group. However, it would seem that more children of high I.Q. and social station are brought for assessment of reading and writing difficulties because of the greater importance placed on education by their parents.

Many theories concerning the pathology have been proposed. Brain's proposal is probably closest to the truth — there is incomplete development of the speech

pathways, leaving the child without normal hemisphere dominance. Thus, the confusion in laterality (i.e. — failure to establish a dominant hand) found so often in association with maturational lag is a symptom rather than a cause of the syndrome.[5]

Conclusion

In conclusion, one might mention a few words about treatment and prognosis.

With dysphasia, the prognosis is very favourable, most acquiring intelligible speech by 6 to 7 years and normal speech by 9 to 10. Development passes through the normal stages, improvement in comprehension occurring first, in articulation last. Some may later show stammering, dyslexia and/or dysgraphia. These children, though their articulation and sentence structure is normal, often have difficulty in expressing themselves verbally for the rest of their lives. It is questionable whether speech therapy does any good; it is usually advised as a form of reassurance for the parents who find the "no treatment" approach difficult to accept.

For dyslexia and dysgraphia, some form of treatment is indicated. The need for special classes is just being recognized and in many centres, the Board of Education provides facilities for those with "learning disorders". In these cases, the aim is not to cure the child of his defect but to provide an alternate method to bypass his learning block. The more seriously affected children may remain in these classes throughout primary school while for others 6 to 12 months of intensive teaching may be adequate.

One of the important facets of the program is establishment of left-right orientation. With extensive facilities, calisthenics to develop the natural movement from left to right, necessary in our right-handed society, are excellent. However, in the ordinary smaller class this is impractical. The child should be taught various devices for distinguishing left from right such as a mark on his hand. These should never be used in place of reasoning, e.g. — figuring out which shoe is for the right foot by the curvature of the sole.

A kinesthetic approach to reading and writing is employed — i.e., tracing with whole arm movement in paint, sand or water the letters or words as he says them, hears them, and sees them written. This involvement of the *three* senses — sight, hearing, and touch — has been found to help in remembering symbols. No printing is permitted; rather, the child is taugh to write, even in Grade 1. This helps maintain the left to right flow and avoids many of the letter reversals. It is often difficult to persuade the teacher to allow the child to continue writing in the primary grades as it is not normally introduced until Grade 3 or 4! Errors within the word are never erased; instead the whole word is rewritten. The reasoning behind this is: if he writes "cake" as "cace", then erases the "c" and inserts a "k", he really learns "cake" as "cacek"!

Reading is taught by the phonetic method — that is, each letter or combination of letters (e.g. "ight") has a certain sound and all words can be "sounded out". This method, unfortunately, is of little use if a defect in auditory comprehension is present; in these cases one usually concentrates on writing and word recognition. A child with reading and writing difficulties makes much better progress in school if he can be given oral examinations rather than written.

The prognosis varies, of course, with the severity. Many children show a striking improvement around puberty but this is unpredictable. Those that do learn to read and write are usually quite slow and have acquired such a distaste for it that they are best encouraged in fields which do not require extensive reading and essay writing.

I think that in considering any form of treatment for these problems it is important to keep in mind that "the acquisition of various skills is dependent on maturation of the nervous system. No amount of practice can make a child sit, walk, talk or acquire other skills until the nervous system is ready for it".[6]

ACKNOWLEDGEMENT

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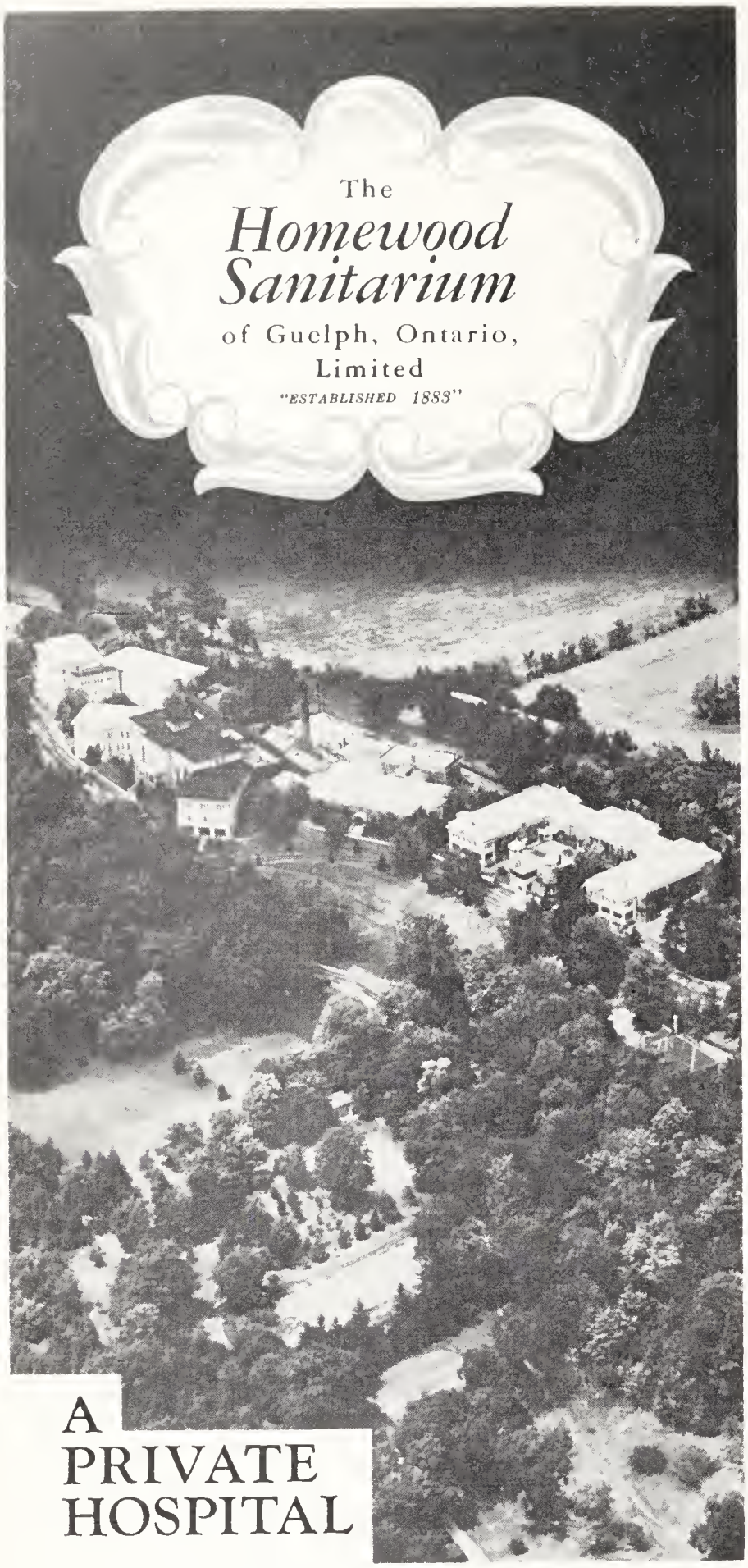

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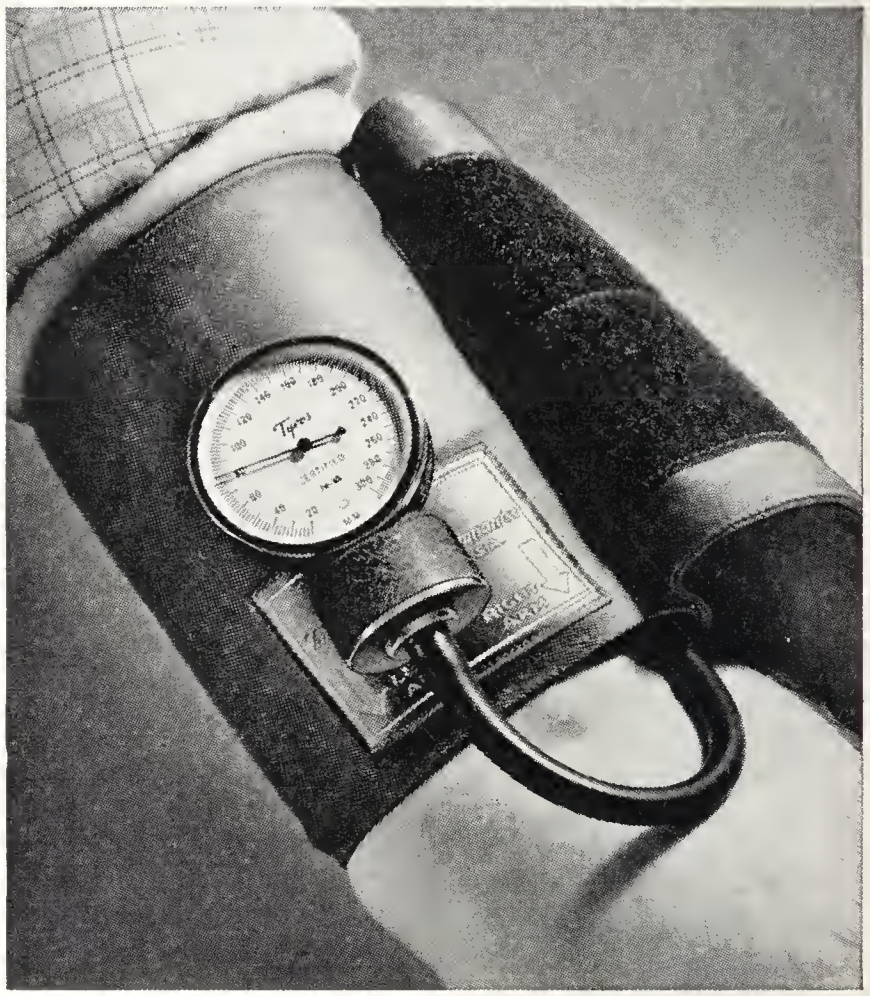

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healed sufficiently, and thereafter, a Taylor caliper may be employed to protect the second hip.

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Rarely, surgery is necessary in order to remove dead tissue, to reshape part of the femoral head, or to relieve the problems of an arthritic joint in later life.

Prognosis

Generally, the older the child at the time of onset of the disease, the poorer the prognosis. The duration of symptoms does not always indicate the stage to which the disease has progressed, as shown radiographically. In most favorable cases, the pain disappears, the femoral head is restored almost perfectly to normal, there is little or no impairment of femoral neck growth, and there is minimal atrophy of the thigh muscles. A period of about 28 months is required for complete regeneration of the femoral head in cases diagnosed early; however, 3 to 5 years are required if the disease is not diagnosed until the stage of fragmentation is reached. Most forms of treatment appear to produce equally good results, but, no form of treatment can correct a flattened femoral head and broadened femoral neck if these are present at the start of treatment. Therefore, as a consequence of late diagnosis or lack of treatment, the patient may be left with a crippled hip. In bilateral disease, the second side is usually recognized earlier, treated sooner, requires less time to heal, and gives rise to less residual deformity than the first side.

Once healed, the affected joint is prone to osteochondritis dissecans, and in later (adult) life to osteoarthritis (due to added stress from joint surface incongruities).

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Book Review

PRESENTING SYMPTOMS IN CHILDHOOD

John Fry, 175 pp. Butterworth and Co. (Canada) Ltd., Toronto, 1962.

This is a book which is written for the general practitioner and the senior medical student. The book is divided into 24 sections, each of which deals with one of the commoner presenting symptoms of childhood illness. The symptoms have been chosen from the author's experience in general practice. Many of the chapters discuss symptoms concerned with respiratory and gastro-intestinal infections.

Essentially, this book presents a diagnostic approach to childhood diseases. A classification of the causes of a symptom, is followed by its pathology, diagnosis and significance. Then, the common conditions causing the symptom are briefly described, followed by a tabulated summary.

This book is carefully and logically organized and is quite readable. Without doubt it could be a useful asset to the general practitioner.

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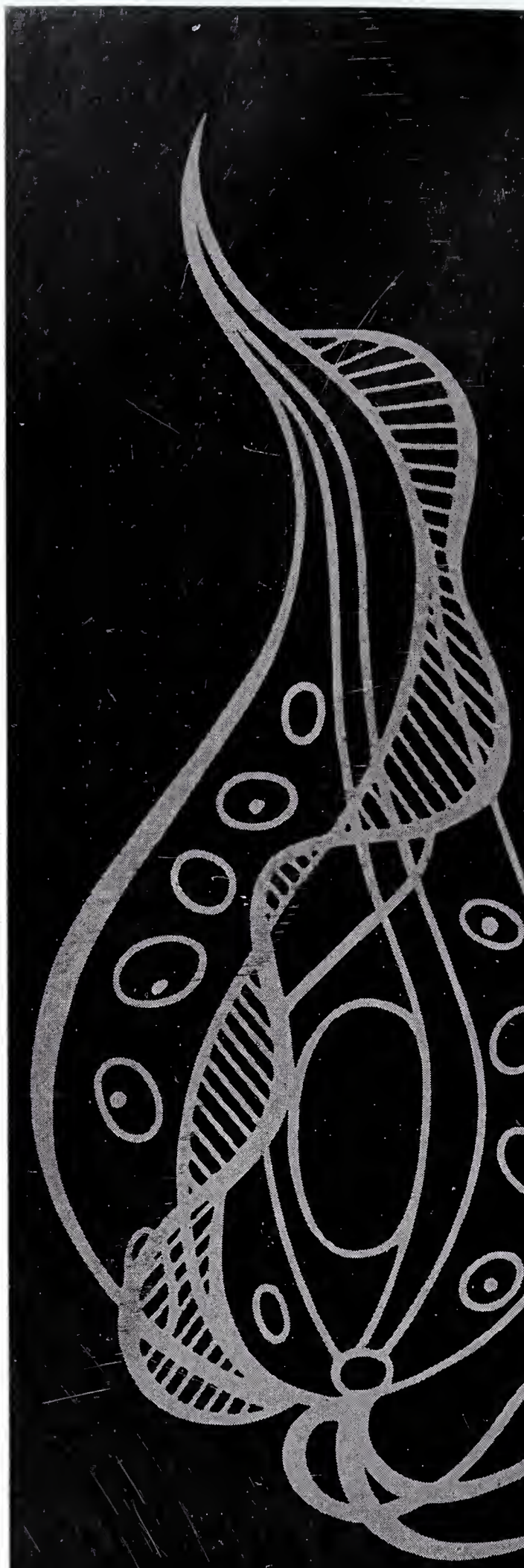

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